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XXIX

REMARKS BY THE PRESIDENT

LOUIS H. CLERF, M.D.
PHILADELPHIA, PA.

We are assembled this morning in the romantic city of New Orleans in response to the call of our Council for the Seventy-Fourth Annual Meeting of the American Laryngological Association.

I am not unmindful of the high honor done me by my election to the Presidency of this Association. I wish to take this opportunity to thank you, the Fellows, for the vote of confidence and regard which places me in this enviable position.

This is our first meeting in New Orleans and our second in the Southland. It is indeed fortunate that New Orleans was selected for our annual meeting place in 1953, since this year will be celebrated the sesquicentennial of the Louisiana Purchase. While it was the desire of the United States Government in 1803 to purchase only New Orleans to insure control of the mouth of the Mississippi,

Napoleon was insistent on giving us a bargain and selling the whole colony without reservation and this he did. The advantages that have accrued from this acquisition are very evident in every respect. This becomes even more apparent if we consult the roster of Fellows of this Association.

Dr. Arthur W. deRoaldes, who was an eminent laryngologist in this city, became a Fellow of our Association in 1893. In 1907, the year of his Presidency, he contributed a sum of money to establish an award for outstanding accomplishments and contributions in the field of rhinology and laryngology. Now known as the "deRoaldes Award" it represents the highest honor that this association can confer.

Dr. deRoaldes was an outstanding teacher, a man of fine character and of great ability. By these attributes and high professional attainments he exerted a profound influence on laryngology, particularly in this area, and aided many of the younger men. One of these, Dr. Robert Clyde Lynch, heeded the old adage, namely, to hitch his wagon to a star, and became associated with Dr. deRoaldes in 1904. He attained Fellowship in the American Laryngological Association in 1915.

Although Dr. Lynch's life came to an untimely end in 1931, he accomplished much in his chosen profession. Those who met him at medical meetings, in his clinics, and in the classroom will remember him as a clear, well grounded, forceful, and inspiring teacher. His successors are carrying on in the old traditions under his inspiration.

The contributions to laryngology of these Fellows and of their contemporaries and successors represent in essence the history and traditions of our Association. We cannot hope to carry on today because of their accomplishments of yesterday; however, they afford a source of inspiration. We must build today with the materials that are available now if we wish to retain the prestige and the distinction that has been, and should continue to be that of our Association.

I should like, at this time, to address my remarks particularly to the younger group of Fellows for we must look to them to carry on the tradition of this Association and to supply leadership. I find that about 84 per cent of our Fellowships either have been or are presently on a faculty of an undergraduate or a graduate school of medicine. This does not imply that a place on a faculty of a medi-

cal school is necessarily a requisite for Fellowship. It does indicate, however, that by and large, the teacher or investigator more often gains recognition. I would recommend, therefore, that the younger Fellows take advantage of every opportunity to teach.

Teaching of medicine is more than an art. The true aim of every one who aspires to be a teacher should be not to impart his own opinions but to enkindle the minds of his students. While certain qualities that go to make a good teacher may be inherited much more can be gained by association, observation, and hard work. A teacher need not be a prolific writer nor a scientific investigator. The latter possesses an advantage, however, for, in addition to imparting knowledge, he arouses in young men certain qualities that he himself possesses, namely a desire for exploration and for discovery in pursuit of nature's hidden secrets. Whoever attempts to teach without inspiring the student to learn is hammering on cold iron.

I believe the successful teacher is one who is familiar with his subject, is able to present it in an interesting and constructive manner and conveys the impression that his every effort and purpose is to impart knowledge to his students. Above all, he must not create the impression that he possesses superior knowledge or ability in a particular subject that the student need not, in fact, could not expect to attain.

An important activity in the development of the laryngologist and the teacher is attendance at medical meetings with participation in the deliberations. Here one will find an opportunity to weigh the character and often the abilities of others. It also affords others an opportunity to make an estimate of him.

I vividly recall one of my early presentations at a meeting of a national medical society. In response to an inquiry by my teacher, Dr. Chevalier Jackson, concerning the paper, I replied that I was not pleased with my presentation. His statement that he never had been satisfied with any of his presentations was most impressive and thought provoking. It emphasized certain fundamental rules in the preparation of lectures and papers, namely, that one should carefully prepare material to be presented and reflect on the method of presentation and, secondly, one should view the presentation in retrospect and provide remedies for one's shortcomings.

I have found it useful to attend meetings in other fields of medicine merely to observe the approach and method of handling the subject by the speaker. One should not imitate others even though it is said to be flattering. It is far better to develop one's own approach. Training, practice, and observation of others may not lead to perfection but they surely will compensate for early inadequacies.

One does not so often respect men in medicine for the abundant possessions they may have accumulated but for their contributions to clinical medicine, research and medical education. The desire to amass a fortune in the practice of medicine often deprives one of certain joys and may blunt one's moral sensibilities. It often seriously impairs one's usefulness in our profession.

May I therefore recommend that, in the interests of our profession, we, the Fellows of this Association, maintain a lively and active interest in the clinical practice of our specialty. In addition, we should be in the forefront in teaching and disseminating medical knowledge.

1530 Locust St.

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PRIMARY PLASMA CELL TUMORS OF THE UPPER AIR PASSAGES WITH PARTICULAR REFERENCE TO INVOLVEMENT OF THE MAXILLARY SINUS

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Primary plasma cell tumors involving the upper air passages are of interest not only because of their rarity but also because of the puzzling and unpredictable behavior which sets them apart from other neoplasms encountered in this area. Although reports of approximately 175 cases have gradually accumulated since the original description by Schridde in 1905, many of these are lacking in critical value because of incomplete clinical and follow-up information. Stout and Kenney[1] for such reasons were compelled to discard 50 of the series of 104 cases which they assembled for analysis from published reports. The evolution of these tumors is characteristically slow and widely varied. Periodic observations over a period of many years are necessary for an accurate appraisal of their true nature and behavior.

THE PLASMA CELL

The plasma cell which forms the basis for these tumors is commonly associated with many inflammatory conditions and shows a predilection for the conjunctiva, alimentary and respiratory systems. Granulomas and polyps of the mouth and upper air passages frequently contain considerable numbers of these cells. Their function is unknown. Most present day opinion agrees that plasma cells are primarily of lymphatic origin, developing as Maximow demonstrated, from lymphocytes. Both plasma cells and lymphocytes are known to be normal although perhaps rare constitutents of bone marrow so that the possible sites of development for plasma cell tumors are

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widespread. The plasma cell presents distinctive characteristics including abundant cytoplasm and a small eccentrically placed nucleus with from five to eight distinct, deeply stained blocks of chromatin arranged in cartwheel fashion.

RELATIONSHIP TO MULTIPLE MYELOMA

The exact relationship of primary plasma cell tumors to multiple myelomatosis has never been fully established. Certainly they have much in common. The age and sex incidence of the two tumors are strikingly similar. The metastatic bone lesions are also alike in distribution and identical roentgenologically as well as pathologically. The life expectancy however, differs markedly in the two groups. Few patients with multiple myeloma survive longer than an average period of two to three years. There are on the contrary many recorded cases of patients with primary plasma tumors of the upper air passages surviving longer than eight years and Oppikofer[2] reported a patient alive and well 31 years after the removal of the original tumors. There are also important laboratory tests which aid in differentiating in nearly two-thirds. Bence-Jones proteinuria can be demonstrated in nearly two-thirds of the cases of multiple myeloma but has never been observed in extramedullary tumors except in the presence of skeletal metastases. A pronounced hyperproteinemia is commonly present in multiple myeloma with a high total serum protein and a reversal of the normal albumin-globulin ratio. Small numbers of myeloma or atyptical plasma cells may also be identified in the blood in over half of these cases according to Sturgis.[3] Furthermore, sternal puncture in cases of multiple myelomatosis will usually demonstrate the presence of typical myeloma cells and roentgenographic examination will show characteristic punched out areas without evidence of bone regeneration especially in the spine, thoracic cage, skull, shoulder and pelvic girdles. It is of interest to note, however, that many authors, including Jackson and Parker[4] feel that there is no sharp line of demarcation between the two conditions, and Ringertz[5] observes that the two groups are undoubtedly closely related although not genetically identical. The cranial lesions of multiple myeloma occasionally involve the jaws and the bony boundary walls of the upper air passages forming tumors within these structures which may be mistaken for primary growths. Nine of the twenty-seven cases reported by Ewing and Foote[6] were of this variety presenting tumors in the nose and throat as the first clinical mani-

festation of what was already a widely disseminated although hitherto silent disease. It is of great importance therefore, when confronted with plasma cell tumors in these areas to rule out the presence of an underlying multiple myelomatosis by appropriate laboratory and roentgenological examinations.

INCIDENCE

All writers on the subject are impressed by the rarity of plasma cell tumors in the upper air passages. New and Childrey[7] observed two such cases among 230 malignant neoplasms of the oropharynx and tonsil. Figi, Broders and Havens[8] found only six cases among nearly 625 malignancies of the nose and sinuses during a 14 year period and only one case among 1,600 cancers of the larynx. Mattick[9] reported two cases among 62,000 admissions to a large clinic for malignant diseases. The number of cases reported in recent years however, has markedly increased indicating a growing awareness of the frequency and significance of the disease in this area.

All writers are similarly agreed on the predilection of these tumors to males after the age of 40 years. In Ringertz's analysis of 47 cases, 80.8 per cent occurred in men between 40 and 70. Geschickter and Copeland[10] reported similar findings. Hellwig[11] observed that the frankly malignant varieties of plasmocytomas tend to occur in a somewhat older group than the so called noncancerous type. Rawson, Eyler and Horn[12] found the average age in the malignant group to be 66 and noted this similarity to multiple myeloma. They found benign lesions occurring in a considerably younger group ranging from 33 to 45. One of the youngest cases observed was reported by Vogt in a 20 year old male with a tumor of the pharynx associated with lymph node metastases.

PATHOLOGIC ANATOMY

Plasmocytomas present a soft, red, lobulated appearance and seldom ulcerate. They occur in three forms as stalked or sessile tumors or as limited areas of submucosal thickening. They vary in size from bean to egg-size dimensions. They usually appear as solitary tumors but are occasionally multiple. Hellwig collected only nine instances of these multiple tumors in his series of 64 cases and Stout and Kenney found 10 such instances in a survey of 104 cases. Blacklock and Macartney[13] reported the presence of five tumors in the nasopharynx and 2 in the larynx in a man of 64. Recent interesting

reports of single cases of multiple tumors have been contributed by Mattick (loc. cit.) and more recently by Priest[14] whose patient, a man of 50, presented multiple recurrent polyp-like tumors involving the nose, nasopharynx, pharynx, and larynx and continued well without evidence of metastases after a period of at least 15 years from the onset of the original lesion. The stalked variety of neoplasm is most commonly observed in the pharynx and larynx but the other types may also occur in any portion of the upper air passages. In the nose, the tumor characteristically fills the passage with a bulky mass which tends to extend into the adjacent sinuses. The tumors originating in the maxillary sinus have for the most part been of soft, friable, easily bleeding character with a tendency to destroy the bony antral walls and to invade the adjacent ethmoids and nasal cavity. As such they are indistinguishable from any malignant neoplasm. In both of my own cases I was impressed by the tremendous vascularity of the plasma cell antral tumor. Lumb and Prossor[15] apparently had a similar experience for they note that the vascularity of the plasmocytoma group in this area tends to be greater than in the case of other tumors. The depth to which these tumors invade is problematical but a survey of reported cases suggests that there must be considerable variations in view of the fact that in many instances superficial removal has resulted in apparent cure while in others radical measures have not prevented local recurrence. As Stout and Kenney have pointed out however, superficial spread in the mucous membranes is common and deep invasion of bone uncommon in marked contradistinction to other malignant neoplasms in this area. The gross appearance of these tumors offers no reliable index of their degree of malignancy since many seemingly innocent polyp-like varieties have subsequently developed fatal lesions in glands and bones.

HISTOLOGIC ASPECTS

The plasma cell tumor consists of a solid growth of plasma cells in massive clusters in a delicate stroma of capillaries and separated by thin septa of connective tissue. Wide variations in actual cell structure occur and, as has been repeatedly emphasized, the microscopic examination provides neither a reliable guide to malignancy nor a dependable clue to the future course of the tumor. Recently, Rawson, Eyler and Horn, while emphasizing that malignant plasma cell tumors may not show the usual cytologic characteristics of malignancy, point out that such criteria, if present in considerable

degree, may be regarded as reliable diagnostic evidence. They believe that *the replacement of tissue* by broad sheets of plasma cells arranged in a delicate capillary stroma is definitely indicative of a malignant tumor and that the absence of this pattern makes malignancy unlikely. The general opinion however, is that in most instances neither the gross nor the histologic appearance of these tumors can be relied upon for help in prognosis or treatment as in the case of other neoplasms.

SITES OF INVOLVEMENT

Plasmocytomas may originate in any portion of the upper respiratory tract as well as on the tongue, alveolar process, palate and floor of the mouth. The tumor has also been observed in the mastoid cells. The conjunctiva is a frequent site of a benign form of this tumor and 47 of Hellwig's series of 128 cases were in this area. It appears from the literature that the nose and sinuses are the section of the upper air passages most commonly involved and further that the most malignant varieties of the plasmocytoma occur in this group. The distribution and frequency of these tumors is indicated in Table 1 compiled from five representative series of case reports.

METASTASES

It is well recognized that in a definite but as yet undetermined percentage of primary plasma cell tumors so-called metastatic lesions appear after varying intervals either in the adjacent lymph glands or bony skeleton. In the latter group the distribution and character of the lesions are entirely similar to those seen in multiple myelomatosis and the roentgenograms as well as histological studies are also identical. These so-called metastatic lesions may be found by careful roentgenographic examination at the time or shortly after the discovery of the original tumor but long periods may elapse before evidence of their presence appears. Jackson and Parker report a case followed for eight and one-half years before local recurrence as well as multiple bone lesions were observed. In a case described by Piney and Riach a 12-year interval elapsed before skeletal metastases appeared. On the other hand, Oppikofer's patient survived 31 years and Priest's 15 years without evidence of metastases. In the series of 104 cases reviewed by Stout and Kenney, 54 showed no metastases, 24 showed metastases to cervical nodes and 15 to single or multiple bones. The fact that *pulmonary* metastases have not been reported,

in the opinion of Stout and Kenney, strongly suggests that these skeletal lesions are not true metastases but rather "new lesions of the same type as the primary tumor, the development of which has been incited by a common, undetermined etiological factor." This of course links this group of tumors with multiple myelomata and Jackson and Parker believe that there is no sharp line of demarcation between the two groups. Lumb and Prossor also favor the conception of multifocal origin rather than spread by metastases. Many reported cases have little value in clarifying this situation because of failure to examine periodically for skeletal lesions, to obtain autopsies whenever possible, and above all to include a followup period over many years.

CLINICAL ASPECTS

The symptoms and signs of plasma cell tumors in the upper air passages are entirely similar to those of other tumors more commonly encountered in this area and the true diagnosis is seldom suspected. In the nose and maxillary sinus in particular the clinical picture corresponds to that of any malignant tumor in this region. In the pharynx and larynx the symptoms are mainly those of obstruction, although in a few instances hemorrhage has been the first presenting complaint. The tumors may appear as pedunculated, sessile or diffuse submucosal elevations. True involvement of the cervical lymph glands is decidedly less frequent than might be expected.

Attempts at classification of these tumors have been unsatisfactory because of the difficulty in separating the benign and malignant varieties on the basis of microscopic and gross characteristics as well as the unpredictable tendency of many apparently innocent lesions later to develop locally invasive or metastatic tendencies. For clinical purposes however, they may be divided into three groups: (1) Apparently benign tumors, solitary or multiple, which are confined to soft tissues. Recurrence may be observed in this group, particularly in the multiple tumors, but invasion of underlying bone does not occur. (2) Solitary or multiple tumors which exhibit local malignancy in the form of invasion and destruction of adjacent soft and bony structures as well as a strong tendency to local recurrence. (3) Tumors associated with invasion of the regional lymph nodes or skeletal osteolytic lesions or combinations of both. In the latter group with bony lesions the clinical picture becomes indistinguishable

TABLE 1.

SITES OF INVOLVEMENT IN THE UPPER AIR PASSAGES.

AUTHORS & DATES OF SURVEY	NASAL CAVITY	SINUSES	NASO-PHARYNX	TONSIL & PHARYNX	LARYNX	MULTIPLE
Ringertz (1938)	5	4	0	2	3	0
Hellwig (1943)	27	8	8	9	10	9
Figi, Broders and Havens (1945)	0	6	2	2	1	1
Stout & Kenney (1949)	17	11	20	23	0	6
Ewing & Foote (1952)	6	6	1	3	1	0
Totals	55	35	31	39	15	16

from that of multiple myelomatosis with characteristic pathological and laboratory findings.

PROGNOSIS

A careful survey of the literature strengthens the opinion that the plasmocytoma represents a frankly or potentially malignant tumor. There are, however, many interesting variations in its behavior. Local recurrence for example may not take place for as long as nine years after the primary removal (Jaeger's case recurred nine and one-half years later) or may be noted within a few months or years. In other instances, after years of apparent cure, without local recurrence, secondary foci may suddenly become evident in lymph nodes, internal organs or bones and progress rapidly to a fatal outcome. It is also possible for patients with local recurrences or lymph node metastases to survive in good health for many years without development of bone marrow involvement. Wachter, Oppikofer, Figi,

TABLE 2.—REPORTED PRIMARY PLASMA CELL TUMORS OF THE MAXILLARY SINUS.

AUTHORS & DATE	AGE & SEX	LYMPH NODES	METASTASES	THERAPY	CLINICAL COURSE
Baldenweck and Periot[17] (1933)	?	0	0	Surgery	Recurrence after 5 yrs.; controlled by radium; well 19 months later.
Cooper[18] (1934)	Male 89	0	0	Surgery & Radium	Unknown.
Blumenfeld[19] (1936)	Male 37	0	0	Surgery, Resection Maxilla	Unknown.
Ringertz (1938)	Male 54	+	+ Left femur	Surgery & X-ray	Died 2 weeks later.
Ringertz (1938)	Male 52	+	0	Surgery	Well—5 months later.
Ringertz (1938)	Male 20	+	0	Surgery & Radium	Recurrence in cervical glands 7 months later, died 13 months later. No local recurrence.
Ringertz (1938)	Male 59	0	0	Surgery	Died of hemorrhage—3 months later.
Figi, Broders & Havens (1945)	Female 54	0	+ (skull)	Surgery & Radium	Metastases to skull, ribs, spine and pelvis after 21 months with death 26 months later.
Figi, Broders & Havens (1945)	Female 37	0	0	Surgery & Radium	Died—meningitis 5 months later.
Figi, Broders & Havens (1945)	Female 70	0	0	Surgery & Radium	Died—9 months later.

TABLE 2.—(Continued)

AUTHORS & DATE	AGE & SEX	LYMPH NODES	METASTASES	THERAPY	CLINICAL COURSE
Figi, Broders & Havens (1945)	Male 55	0	0	Surgery & Radium	No recurrence—4 months later.
Figi, Broders & Havens (1945)	Male 55	0	0	Surgery & Radium	Well, without recurrence — 2 years later.
Lumb & Prossor (1948)	Female 24	0	0	Surgery & Radium	Recurrence after 5 years; controlled by further surgery and radium; *well 2 years later.*
Lumb & Prossor (1948)	Male 44	0	0	Surgery & Radium	Recurrence in 3 months; further radium; metastases to glands, ribs and spine 1 year later. Died 3½ years later.
Wille[20] (1948)	Male 55	0	0	Radiation	Dramatic improvement; alive and well 6 years later.
Parkes and Burtoff[21] (1949)	Male 60	0	0	Surgery & X-ray	Well—7 months later.
Fuerste and Zuckerman[22] (1950)	Male 56	+	0	Radiation	No improvement of note. Died 2 years later.
Ewing & Foote (1952)	Male 45			Details not given	Alive—8 months later.
Ewing & Foote (1952)	Male 73	0	0	Details not given	Died—2 years later.

TABLE 2.—(Continued)

AUTHORS & DATE	AGE & SEX	LYMPH NODES	METASTASES	THERAPY	CLINICAL COURSE
Ewing & Foote (1952)	Male 70			Details not given	Alive 2 years later; no recurrence.
Ewing & Foote (1952)	Female 54			Details not given	Alive 9 months later; no recurrence.
Ewing & Foote (1952)	Male 76			Details not given	*Died*, pneumonia, 16 months later.
Rawson, Eyler & Horn (1950)	Male 33	0	0	Surgery only	Well and no recurrence—3 years later.

Table 2.—Summary of available data on all reported cases of primary plasma cell tumors of the maxillary sinus. Cases with secondary invasion from adjacent structures are *not* included. Of the total of 23 patients, 10 died from two weeks to three and one-half years after treatment. Eleven reported living from four months to seven years later. Fate of 2 cases unknown. In most instances treatment consisted of combined surgery and irradiation.

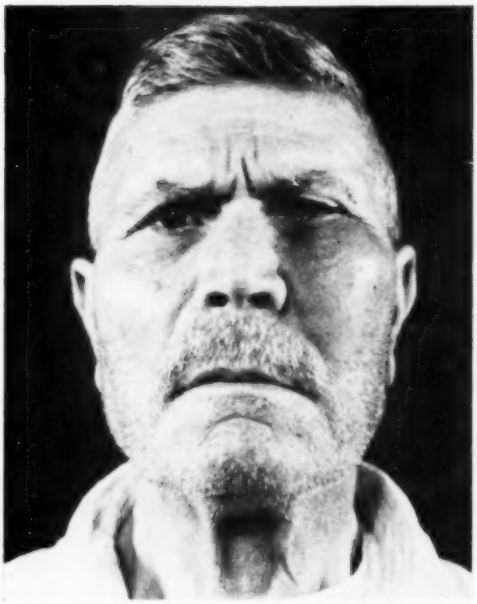

Fig. 1, Case 1.—Plasmocytoma of the left maxillary sinus in a man of seventy.

Stout and Kenney have all reported such patients surviving seven and one-half to thirteen years without difficulty. It may be accepted that the best although necessarily guarded prognosis can be rendered in cases confined and localized to soft tissues and the most unfavorable to those showing local invasion of bone or distant skeletal structures. Tumors of the maxillary sinus in particular appear to exhibit a high degree of malignancy. Stout and Kenney however, report one patient with a primary tumor in th nasopharynx alive and well seven years after multiple osseous metastases were demonstrable and collected three similar cases from the literature surviving from 4 to 10 years. It is evident therefore that the prognosis is decidedly uncertain in these tumors. It should be stressed again that neither the gross nor the microscopic appearance of these neoplasms affords reliable prognostic information and that the histologic features of the

benign granulomatous lesions of the conjunctiva may closely resemble those of the rapidly growing malignant varieties in the air passages. Figi, Broders and Havens conclude their study with the opinion that these tumors should be classified and treated as malignant tumors and the accumulated evidence certainly supports this view.

TREATMENT

The treatment employed in reported cases consists of surgical removal, radiotherapy, or a combination of both methods. The plasma cell as would be anticipated from its close relationship to the lymphocyte is usually radiosensitive although some cases with indifferent response to irradiation have been reported. Stout and Kenney after analyzing the methods of therapy employed in 16 patients surviving more than five years conclude that "since irradiation is less mutilating than surgery and has at least as good, if not better, chance of success as surgery, it would seem to be the treatment of choice in most cases." Cade[16] also considers these tumors to be radiosensitive and advises irradiation. Ewing and Foote report encouraging and occasionally dramatic early responses to irridation in several of their cases including the difficult nose and sinus group accompanied by bone involvement. Rawson, Eyler and Horn also stress the radiosensitivity of these tumors and feel that radiotherapy is the treatment of choice except in the frankly benign group which can be adequately treated by surgical excision. Pedunculated tumors in most reported cases have been removed surgically, their base cauterized or coagulated and subsequent irradiation frequently employed. The combination of surgery with intracavitary radium and deep roentgen therapy seems to be the accepted method of dealing with plasma cell tumors involving the maxillary sinus (Table 2). The tremendous variation in the known response of plasma cell tumors to any type of therapy as well as the strong possibility of subsequent involvement of lymph glands or bone marrow in spite of any known form of local treatment increase the difficulties of thoughtful management. Indeed if such so-called extensions actually represent multiple primary involvements rather than true metastases, the advisability of extreme radical surgical measures in eradicating the original tumor seems questionable. Until more reports are available which include careful follow-up studies over a period of several years, it would seem more in keeping with our present knowledge of this tumor to employ both surgery and irradiation but to avoid needlessly radical procedures.

Fig. 2, Case 1.—Roentgenogram showing a large tumor with extensive destruction of the walls of the left maxilla.

REPORT OF CASES

CASE 1.—A 70-year old Italian was first seen at the Strong Memorial Hospital on October 16, 1946 complaining of progressive swelling of the left side of the face of nine months' duration accompanied by recent intermittent pain (Fig. 1). A firm mass could be palpated over the left maxilla and at one point distinct pulsation could be felt. The roof of the mouth was intact and there was no intranasal evidence of extension. There were no palpable cervical glands. The roentgenograms showed a tumor mass which had destroyed the anterior and the upper walls of the left maxillary sinus and had involved the adjacent malar bone (Fig. 2). The chest films showed evidence of an old apical tuberculosis. A diagnosis of malig-

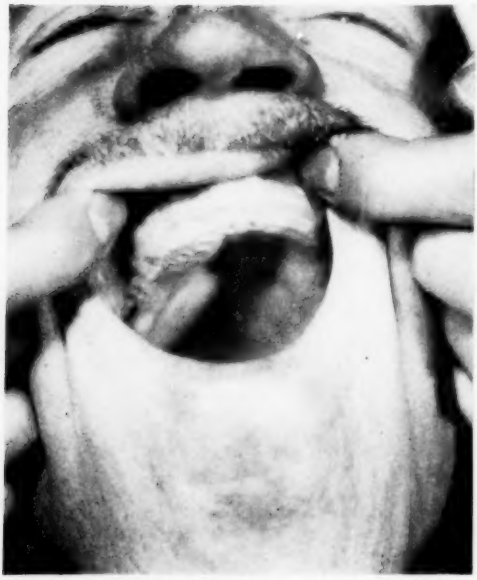

Fig. 3, Case 2.—Palatal swelling, the chief presenting complaint in a man of forty-five.

nant tumor of the left antrum was made and exploration was advised. This was carried out on October 20, 1946, entering the antrum by way of the canine fossa. The sinus cavity was found to be filled with friable tissue which adhered to the bony walls and was tremendously vascular. Attempts to control the bleeding by coagulation and hot packs were unsuccessful and ligation of the external carotid artery was carried out with surprisingly little immediate effect. Finally, however, the bleeding was controlled by firm packing. The patient was supported by transfusion and made a rapid postoperative recovery. The histologic examination of the tissues removed at operation showed a characteristic plasmocytoma. A complete metastatic survey of the bony skeleton showed no roentgenological evidence of metastases. The electrophoretic studies were not suggestive of multiple myeloma. The urine was negative for Bence-Jones protein.

The Wasserman was negative. A sternal puncture was carried out and no myeloma cells were reported. The lesion therefore, was considered to represent a primary plasma cell tumor limited to the left maxillary sinus. Roentgen therapy was instituted delivering 5800R to the tumor through three portals. Following this the soft tissues over the inferior orbital and malar aspects gradually broke down and several sequestra were removed through this large opening. The patient was followed closely and remained in good health until April, 1949 when he began to complain of pain over the lumbar spine. Roentgenograms now showed an early destructive lesion in the first lumbar vertebra. A metastatic survey was again negative except for a small punched out area in the left frontal region. There was now a definitely positive Bence-Jones proteinuria present and a sternal puncture showed an increase in plasma cells. The electrophoretic studies were still not typical of multiple myeloma. The patient's course became progressively worse and death occurred on October 20, 1949, nearly four years from the onset of his facial swelling. An autopsy was carried out and showed bilateral apical pulmonary tuberculosis with miliary tuberculous lesions in the liver, spleen, adrenals and kidneys. Bronchopneumonia was present. The destructive lesion in the first lumbar vertebra was considered to be entirely tuberculous until more careful subsequent restudy showed early plasma cell involvement as well. The internal organs were free of plasma cell metastases and there was no involvement of the cervical glands.

CASE 2.—A white male, age 45, was first seen on February 18, 1936 complaining of chronic right-sided nasal discharge and recent swelling of the roof of the mouth on the right side. Examination showed slight fullness of the right side of the face and moderate swelling of the palate beneath the right antrum (Fig. 3). There were no palpably enlarged regional glands. The right antrum was opaque on transillumination and an attempt to irrigate this sinus met with resistance suggestive of tumor. Roentgenograms reported the right antrum to be completely obliterated by a dense shadow with evidence of thinning and irregularity of the bony floor suggestive of a malignant neoplasm. Blood studies were normal. On March 10, 1936 the right antrum was explored through the canine fossa. The antrum was found to be filled with a friable, adherent, *extremely vascular* tumor which had apparently originated along the floor of the sinus. It was removed by curettage. Microscopic examination revealed a typical plasmocytoma. A complete metastatic

survey was then carried out and found to be negative. There was no Bence-Jones proteinuria. The blood studies and blood chemistry were not altered. Sternal puncture studies were not made. The patient was given intensive Roentgen therapy over the right antrum, receiving 5,600r through multiple portals. The result was clinically satisfactory and he was entirely free of local symptoms except for a small draining tract into the mouth. He was followed at frequent intervals in the Out-Patient Department and remained well until his admission to the hospital on March 18, 1939 after drinking 100 cc of Carbona. A confirmed alcoholic, he had accidentally taken this solution during a bout of drinking. Death occurred on March 27, 1939 due to acute carbon tetrachloride poisoning. Autopsy showed no local recurrence nor evidence of metastatic plasma cell tumor. This patient therefore remained free of clinical or post-mortem evidence of plasmocytoma three years after the diagnosis had been first established.

CONCLUSIONS

1. The controversial and unpredictable behavior of primary plasma cell tumors can be clarified only by careful clinical, laboratory and postmortem studies as well as by continued observation of these cases over long periods. A large number of reported cases possess little critical value because of incomplete follow-up data.

2. The discovery of a plasma cell tumor in the upper air passages requires first of all careful roentgenological as well as laboratory studies to rule out the possibility of multiple myelomatosis. Sternal puncture should be included in this study. The course of this disease is notoriously silent for long periods.

3. The relationship of these tumors to multiple myeloma appears to be a close one although there are characteristic and important differences including a slower progression and the possibility of long survival in certain cases.

4. The increasingly prevalent opinion among pathologists and clinicians is that primary plasma tumors in the upper air passages, whether limited to the soft tissues or also involving bone, are in the vast majority of cases, variants of multiple myelomatosis. In any event, there can be no question about their malignant or potentially malignant character.

5. The methods of management as well as the prognosis must necessarily vary with the individual case. The radiosensitivity of these tumors favors radiotherapy. Experience does not appear to justify the radical surgical procedures commonly employed in the treatment of the more familiar malignant neoplasms encountered in this region.

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XXXI

THE MESENCHYME OF THE NOSE AND SINUSES

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Rarely in the history of medical science has a large structure of the body been neglected for so long a time as has the ground substance of connective tissue. On the other hand, almost never has a large structure gained such sudden and tremendous scientific importance as has recently been given to this supposedly dull and uninteresting amorphous gel.

Why has this structure suddenly developed this great personality, which has drawn so much interest from the medical profession all over the world?

I believe the main reasons to be as follows: (1) In the past, stained preparations of ground substance have shown little, if anything, of interest. Newer techniques now give visible evidence of this structure. (2) The development of electron microscopy has brought detail into connective tissue which previously could only have been hypothesized or imagined. (3) The use of tissue cultures has added much to the knowledge of connective tissue physiology and growth. (4) The advent of ACTH and Cortisone has stimulated great interest in this tissue.

Connective tissue and its elements have a wide and large distribution in the body. Some of the common areas are found submucosally, subcutaneously, surrounding vessels, nerves, and muscle fibers, and in the parenchymatous organs.[1] If the ground substance under the epithelium of the nose and sinuses alone were flattened out into one plate it would make quite a display of gel.

Mesenchyme is embryonic connective tissue and is the precursor of adult connective tissue. Mesenchyme is a loose gel, consisting mostly of ground substance with some small cells and a few fine

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branching fibrils.[1] Wharton's jelly of the umbilical cord is a good example of this structure. The tissue from a nasal polyp or from an allergic nose resembles mesenchyme. The ground substance of any loose, adult connective tissue is practically the same as mesenchyme and is often so designated.

Adult connective tissue consists of this homogeneous matrix in which are incorporated certain fibers and cells.[2] The tissue may be loose or firm, depending upon the relative amounts of these constituents.[1] Loose connective tissue contains more ground substance, dense connective tissue contains more fibers. The connective tissue in the nose is of the loose type.

Cellular elements in connective tissue consist mostly of fibroblasts. Other cells of importance are macrophages, mast cells, eosinophils, and plasma cells.[1]

The fibrillar elements consist mostly of collagen fibrils and fibers. The fine fibrils develop from fibroblasts and are characterized by having crossed striations placed at regular intervals. These fibrils become matted together as in a cable, to form adult collagen fibers (Fig. 1). Collagen makes up about forty percent of the mass of the body.

The ground substance is often referred to as amorphous substance, gel, mucopolysaccharides, or mesenchyme. As stated previously, this structure is a gel-like homogeneous mass and is made up chemically of a combination of carbohydrate and protein.[1, 3] The carbohydrate part is composed of polysaccharides. The main polysaccharide involved is hyaluronic acid. The mucopolysaccharide combination is polymerized.[4] By polymerized, we mean that the structure is made up of small aggregates. The more highly polymerized it is, the firmer it is. However, this gel may be broken down, depolymerized, or made less firm by enzyme activity. The main enzyme involved is hyaluronidase. Collagenase may also be of importance in this regard. The gel is maintained and built up by enzymatic activity of the fibroblasts. Hence, the consistency of the ground substance may vary from a firm gel to a more liquid or soluble state depending upon the activity of the enzymes. Existence of hyaluronidase and other enzymes in some of the mesenchymal cells has to be postulated if maintenance of the amorphous substance is to be explained.[14] Like all structures in the body this gel is constantly being built up and broken down. Therefore the enzymes must be present in the substitute in large

Fig. 1.—Connective tissue fibrils seemingly developing from the surface of a fibroblast. Reprinted by permission from Ralph W. G. Wyckoff: The Fine Structure of Connective Tissues. Trasactions of the Third Conference on Connective Tissues. Josiah Macy, Jr. Foundation, Publishers. (Feb. 14-15) 1952.

amounts. Hence they must be present in inactive or bound form ready to be activated as needed. Mayer[17] has shown that in allergic and non-allergic skin inflammations there is a forty fold increase in local hyaluronidase. Since these inflammations can be reduced by neutralizing the hyaluronidase Mayer concludes that this enzyme participates actively in inflammatory reactions.

The extracellular tissue fluid is incorporated in the ground substance and comes from the blood plasma, containing water soluble proteins, metabolites, crystalloids, gases, et cetera. In normal connective tissue, this tissue fluid is in bound form and is not free.[1] In other words, if one should stick a knife into the amorphous sub-

stance of a normal turbinate, no fluid would run out. However, in any inflammatory edema, free fluid is present.

In the past, about the only function given to the ground substance was to hold the body together and give it a certain amount of mobility. Let us see whether or not this structure has other duties.

After an infectious organism passes the surface epithelium, the ground substance is the first structure it encounters. Hence, the importance of this gel as a barrier against the spread of infection must not be overlooked. In general, a normal firm gel acts as a good barrier against the spread of an ordinary infection. On the other hand, a more liquid gel aids in the spread of most infectious organisms. However, a condition known as the colonial effort must be taken into consideration.[5] It takes a certain number of infectious organisms to infect an animal or to grow in a given amount of medium. If only a few weak or nonvirulent bacteria are placed in a large amount of medium, they will die out. A larger number of the same organisms in the same amount of medium will survive. If the organisms are stronger or more virulent, just a few will survive in this same large amount of medium. This is also true in the animal body. This shows that there is a definite relationship between the quantity of infectious agent and the amount of material in which it is grown. This ratio is definite for each organism. This is known as the colonial effect and it applies to the ground substance. In other words, hyaluronidase, which liquifies and increases the amorphous substance, will enhance the activity of organisms of high virulence while the dilution effect of the same enzyme will suppress the growth of nonvirulent organisms. Hence, in some cases the host and the condition of the ground substance may be of more importance than the invading organism. This may help to explain why nothing serious happens to patient A when he catches a cold or goes swimming, but under the same conditions, patient B develops an acute and fulminating pansinusitis.

Another phenomenon of importance in this regard is the fact that many streptococci and pneumococci, as well as some staphylococci, secrete hyaluronidase.[5] This produces even further invasiveness and more damage from these virulent organisms. For example, in cases of pneumococcus pneumonia which develop bacteremia, the pneumococcus produces excessive amounts of the spreading factor.

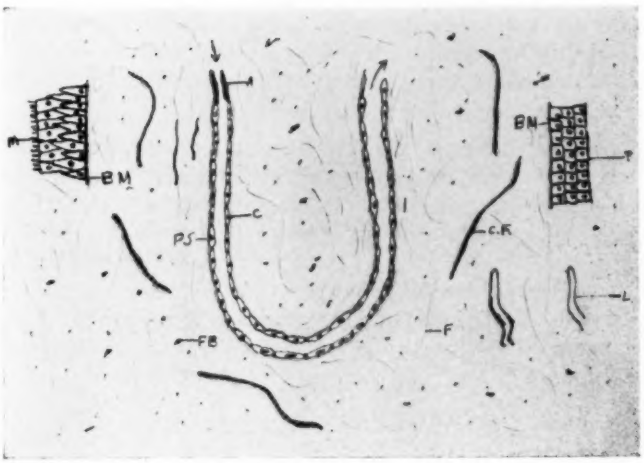

Fig. 2.—A: Arteriole; C: Capillary; F: Fibril; L: Lymphatic; M: Nasal mucosa; P: Parenchyme; BM: Basement membrane; CF: Collagen Fiber; FB: Fibroblast; PS: Peri-capillary sheath.

In uncomplicated cases of pneumococcus pneumonia, there is less production of this enzyme by the organism.[6] This shows that both the host and the invader participate in producing the difference in the hypothetical cases of patients A and B.

As usual in such situations, nature has not let us down. The body also develops anti-spreading factors, or anti-hyaluronidases.[7, 2] These inhibitors may be demonstrated in the blood-stream. There is a specific antihyaluronidase and a non-specific inhibitor. For example, in many streptococcus infections, a specific antibody is built up to overcome the spreading factor produced by the streptococcus. In cases of acute rheumatic fever this antibody is very high. The non-specific inhibitor is not an antibody and its exact nature is not known. Recent work indicates that it may be heparin or a derivative thereof.[2, 8] My personal feeling is that it is in some way connected with the stress hormones of the adrenal cortex. This non-specific anti-hyaluronidase is high in the acute stages of all connective tissue disease and in fact, in most acute infections and in other

conditions of stress and strain.[2] Hence, the common denominator which is necessary for the production of this substance is a stimulation of adrenal cortex by some stress.[9] This statement can be proven by the experiments in which normal animals under stress produce a high blood level of the non-specific inhibitor, while stressed adrenalectomized animals do not.[9]

The ground substance also has certain growth functions. It has been shown that around the edges of rapidly growing tumors, the gel is liquefied (probably by hyaluronidase activity) thus enabling the tumor to spread and grow more readily.[10] This can be determined by the use of intravital staining. For example, Evans Blue has a selective staining action on depolymerized ground substance. If this dye is injected intravenously into animals with rapidly growing tumors, it shows up in the ground substance near the edges of these tumors.[10]

A similar process may take place in regard to metastases. Gersh and Catchpole[10] believe that metastatic tumors live and survive where the ground substance is favorable. They believe that the metastatic cells may stimulate the secretion of depolymerizing enzymes which produce a favorable site in the ground substance.

How does material get from the blood-stream to the cell? It must go through the capillary wall, the pericapillary membrane, the ground substance, and the cell membrane.[1] Substances from the cell get back into the circulation in the reverse manner. Again the consistency of the ground substance will help to determine this process. Undoubtedly, this is a function of this structure (Fig. 2).

While regular enzymatic activity maintains the connective tissue elements, certain vitamins and hormones participate in speeding up or inhibiting these reactions. Ascorbic acid is the most important vitamin in this regard.[2] Vitamin C is very high in the adrenal cortex, and when the body is under stress the cortex is quickly depleted of this vitamin. In some way, ascorbic acid is a factor in the production of the stress hormone.[11, 12] In vitamin C deficiency, there is a failure to produce the polysaccharides and therefore, a poor ground substance results.[2] Also, in this deficiency, new collagen fibers are not formed, although the old fibers will be fully maintained without ascorbic acid.[2] Thus, scurvy is associated with poor healing.

Various of the hormones are important in this picture.[2] Thyrotropic hormone stimulates the production of ground substance and

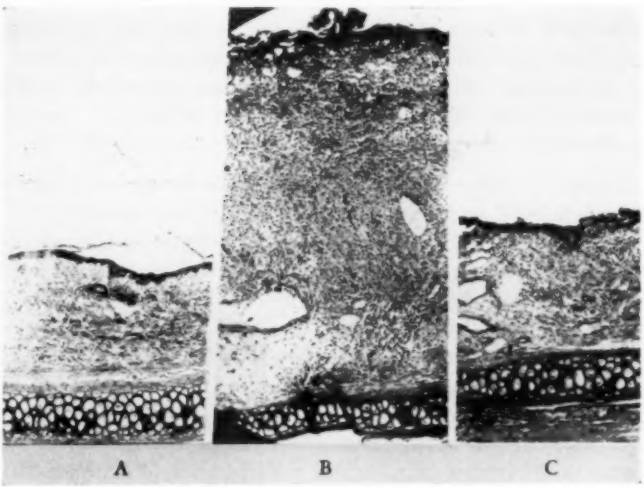

Fig. 3.—A—Cross section of normal rabbit's ear—left. B—8 day granulating wound control—center. C—8 day granulating wound, animal treated with cortisone—right. Note difference in thickness in granulation tissue in control and animal treated with cortisone. Reprinted by permission, from Bull. N. Y. Acad. Med. 25:252.

collagen. For example, if a thyroidectomized guinea pig is given thyrotropic hormone exophthalmos is produced from an excessive production of retrobulbar gel.[2] Estrogens decrease the permeability of amorphous substance while gonadotropins increase it.[13] Cortisone decreases the permeability of the ground substance while desoxycortisterone increases it. Cortisone depresses the production of all of the elements of connective tissue, namely, the ground substance, collagen, fibroblasts, and capillaries.[14] Therefore, this hormone may inhibit wound healing[18] (Fig. 3).

Edema is the accumulation of excessive fluid in the intercellular spaces, i.e. in the ground substance. For example, Reinhart's special staining technique has shown that allergic reactions involving the respiratory tract produce edema in the mucopolysaccharides of the nose and the bronchial tree.[15] The mechanism of the production of edema is somewhat in question. Time does not permit a complete

discussion of the pros and cons of this subject. However, to put it briefly, there are two schools of thought: One believes that edema is due entirely to some direct action on the intercellular cement of the capillary endothelium. The other believes that the edema may be due to liquefaction of the ground substance around the capillaries thus loosening endothelial connections from without.[16] The action of hyaluronidase on ground substance will apparently do this.

Connective tissue damage of any kind is always associated with an increase as well as a liquefaction of the ground substance.[1, 2] In fact this liquefaction, or depolymerization, may make the amorphous substance so soluble that it passes into the blood-stream where it can be found and measured.[2] Therefore, connective tissue damage is associated with, or results in, the following: (1) edema in the ground substance, (2) increase in the ground substance, (3) seepage of the ground substance into the circulation, and (4) increase in the non-specific antihyaluronidase in the blood. The damage may be of an allergic or an infectious nature, or from any stress. If this stress continues long enough in a certain type of individual, then further degenerative processes may take place. Precipitates may be deposited in the ground substance producing amyloid degeneration, hyaline degeneration, and so forth. The ultimate end result will be more collagen and less ground substance with eventual sclerosis.[2] This process is probably one of the reasons why some individuals grow old prematurely.

There are many other interesting points that can be made in reference to the ground substance of the nose and sinuses, but lack of time and lack of knowledge eliminate further observations.

SUMMARY AND CONCLUSIONS

The development and the morphology of the mesenchymatous ground substance has been pointed out. The possibility of new and interesting functions of this structure has been presented. The enzymatic and hormonal control of the mesenchyme has been reviewed. The importance of the ground substance in allergic and non-allergic inflammations of the respiratory tract has been stressed. A brief resume of connective tissue pathology from edema to sclerosis has been given.

The intent has been to place the ground substance of the respiratory tract in the position of a structure of some importance, and

not merely as an inert tissue whose single duty is to hold the anatomical structures together.

384 Post St.

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XXXII

PRIMARY AMYLOIDOSIS OF THE LARYNX

WITH A REPORT OF EXPERIMENTAL TREATMENT WITH CORTISONE

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AND

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During the past three years we have observed four cases of so-called primary or idiopathic amyloidosis of the larynx. This condition is of sufficient interest, despite its rarity, that it seems worth while to present a brief review of current opinion about this type of lesion and to note the direction of research studies now being carried out in relation to amyloid deposits in various tissues of the body, along with a report of a clinical trial with cortisone in two of our own cases and with ACTH in a case observed by Dr. Irl Blaisdell, of Syracuse, New York.

INCIDENCE

The reported incidence of primary amyloidosis either of generalized type or in solitary sites such as the larynx, is very low. For example, Dillon and Evans[1] reported that only three cases of primary amyloid disease were observed at Peter Bent Brigham Hospital in Boston during a twenty-eight year period from 1913 to 1941, which included 120,785 admissions and 41,551 autopsies; and in none of the three cases was the lesion situated in the larynx. A very recent communication from DeGraf Woodman[2] informed me that he had just observed a patient with primary amyloid tumor of the larynx which was the second such case ever recorded at Presbyterian Hospital in New York.

In our Cleveland hospitals, it would appear that the incidence of amyloidosis of the larynx is considerably higher. Besides the four cases we have seen since 1950, thirteen cases of amyloid disease of the

larynx have been recorded at another hospital during the last fifteen years. This experience is similar to that reported, in 1949, by Kreissl, Muirhead and Darrough,[3] of Dallas, Texas. They reported five cases of primary amyloidosis of the larynx seen in a period of twenty-four months in a surgical pathology service associated with two laryngology services; during this period, specimens from thirty-one laryngeal lesions were examined. Beavis,[4] in 1934, reported five cases he had observed and commented that amyloidosis of the larynx is probably not so rare as the paucity of reports in the literature would seem to indicate.

Whether the apparently higher incidence of primary amyloidosis of the larynx in some places, such as Cleveland and Dallas, is due to greater interest on the part of the pathologists in carrying out special staining techniques for the recognition of amyloid, or whether there is some geographical, nutritional or environmental factor which causes an actually increased incidence in certain places cannot be answered now. However, it seems likely that if amyloid disease of the larynx were looked for more systematically by both clinicians and pathologists, there might be an increase in the reported incidence.

CLASSIFICATION

The association of amyloid deposits in various tissues of the body in the presence of chronic wasting diseases, such as tuberculosis and syphilis, has long been recognized as a secondary degenerative process, in which the organs most usually affected are the spleen, liver, kidneys and adrenal glands. There is also a type of so-called primary systemic amyloidosis, in which there is no demonstrable pre-existing infection, and the amyloid deposits are found in the heart and organs other than those usually involved in secondary amyloidosis. Besides this generalized type of primary amyloidosis, there are the solitary amyloid tumors situated in various sites, but particularly in the larynx and upper respiratory tract, which are also regarded as primary lesions, since they occur without any demonstrable pre-existing chronic infection.

The foregoing is an oversimplified statement of the principal categories of amyloid disease. Actually, the cases show many deviations from any such categories, and the elaborate classifications that have been proposed by some writers have served only to confuse the picture. Since there are still so many questions to be answered re-

garding amyloid deposits in the body and their localization, the trend at present is to simplify the basis of classification.

King,[5] in 1948, registered an objection to the terms, primary and secondary amyloid disease, suggesting that when the basic mechanism is known, all cases will be found to be of secondary type. King proposed a classification in which the main differentiation is based on the so-called typical or atypical distribution. Whittlesey,[6] in 1950, suggested reversing these headings, that is, basing the principal differentiation on whether or not the amyloidosis is associated with some other disease, rather than on its distribution in the body. His scheme is as follows:

1. Primary amyloidosis (not associated with a disease)
    a. Typical distribution
    b. Atypical distribution
2. Secondary amyloidosis (associated with a disease)
    a. Typical distribution
    b. Atypical distribution

The classification proposed by Dahlin,[7] is the following:

1. Primary amyloidosis
    a. Systemic
    b. Focal or tumefactive
2. Amyloidosis complicating myeloma
    a. Systemic
    b. Focal or tumefactive
3. Secondary amyloidosis

According to the classification of Dahlin, the cases of laryngeal amyloidosis that we have observed are of the primary type, with focal distribution, since no evidence of amyloidosis elsewhere in the body could be adduced. It must be stressed, however, that this classification is a clinical one and, as such, is valid only for the time during which the patients have been under observation. Future findings might indicate the presence of amyloid deposits in other organs of the body, as in the case cited by DeWolf and Clarke,[8] in a man aged 59, with clinical symptoms simulating carcinoma of the larynx, carcinoma or ulcer of the stomach, rheumatic endocarditis and adhesive

pericarditis. This patient's first symptom had been hoarseness present for four or five years before he sought medical help for the relief of gastric symptoms. Throughout the two-year observation period, the laryngeal symptoms became more severe, and biopsy confirmed the diagnosis of amyloidosis. Necropsy showed excessive deposits of amyloid in the tongue, larynx, myocardium and cardiac valves, submucosa and muscularis of the gastro-intestinal tract, lymph nodes, urinary bladder, mesentery and beneath the serous surfaces. On reading the literature, it is impossible to determine whether laryngeal involvement was present in the cases which have been reported as primary systemic amyloidosis, since most of the authors failed to mention the larynx which is not routinely examined *post mortem* in most institutions.

Although the supposedly primary solitary amyloid tumor of the larynx no doubt is accompanied by disseminated amyloidosis in some cases in which the generalized condition is not recognized, there is good evidence that many of these cases are indeed solitary, localized lesions. The separation of the local nodular deposits from the systemic or disseminated deposition is not necessarily clear-cut, but so far, according to Kreissl and his associates,[3] the reports of the focal form have exceeded the cases of the systemic form by at least a factor of 2. Stark and New[9] and others have called attention to the fact that amyloidosis of the larynx tends to be self-limiting, another factor which would seem to indicate that it is a localized and not a generalized progressing condition.

SYMPTOMS

The symptoms due to amyloidosis of the larynx are caused by mechanical factors and hence are conditioned by its size and location. If there is no infringement of the vocal cords or obstruction of the airway, the lesion is asymptomatic and may be discovered only on routine examination. Hoarseness of several months' duration is the principal symtom, but dyspnea may also be present in the case of larger lesions. According to Stark and New,[9] men are more frequently affected than women in a ratio of two or three to one, and the disease occurs most commonly in the fifth, sixth and seventh decades of life. Two of our patients were young women in their early thirties, and it is our impression from this experience and from reading the literature that primary amyloidosis of the larynx tends to occur at an earlier age in women than in men.

Fig. 1.—Low-power view of the laryngeal mucosa stained with hematoxylin-eosin. Note that in the center of the field there is a hyaline-like tissue arranged in sparsely nucleated broad, irregularly anastomosing bands. Some of this tissue change is particularly apparent around a small blood vessel in the center of the section. The degenerative changes are more obviously demonstrated at a higher magnification with this type of stain; they are brought out by selective staining in Fig. 2.

CLINICAL FINDINGS

Amyloidosis of the larynx may appear as a local tumor, but often, the lesion is not well localized and may be described as "diffuse submucosal thickening." The surface may be smooth or nodular, but characteristically the superficial mucosa is healthy. Clinically, the absence of ulceration of the mucous membrane is the most significant observation leading to the suspicion of amyloid tumor. In some instances, the lesion has a definite yellowish color, as has been noted by numerous authors. In other instances, the laryngeal swelling has a reddish, hyperemic appearance. The diagnosis is made by microscopic examination of stained sections of tissue removed for biopsy.

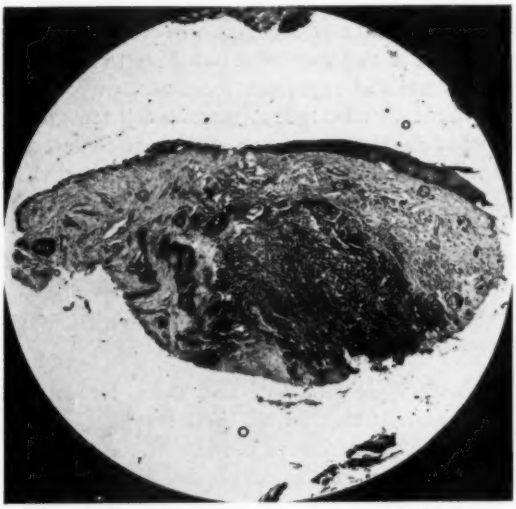

Fig. 2.—Low-power view of laryngeal mucosa in a field comparable to that of Fig. 1. Crystal violet stain shows the metachromasia of the tissue which is the seat of amyloid degeneration. The distinctive staining of the amyloid is well demonstrated in the broad bands of purple-staining tissue in the walls of the blood vessels and in the collagen which occupies most of the center of the section.

PATHOLOGIC FINDINGS

In microscopic sections of tissue removed from the larynx, amyloid deposits are seen as a homogeneous material which is revealed by staining with eosin, crystal violet and methyl green. Figure 1 shows the appearance of amyloid deposits in a section of laryngeal mucosa stained with hematoxylin-eosin and Figure 2 a comparable field stained with crystal violet.

Stark and McDonald[10] reported a histologic study of fifteen cases of amyloid tumor of the larynx, trachea and bronchi, in 1948. Although their study was limited to the "tumor"-forming amyloid lesions, they found no significant difference microscopically in these and in the diffuse lesions. These authors excluded seven cases which had previously been diagnosed as amyloid tumor. In these cases the

lesions were considered to be caused by hyalinized fibrin or tissue exudate identical to lesions described by Huebschmann, who regarded them as an early stage of formation of amyloid. Although Stark and McDonald could not determine whether the pathologic findings in these seven cases did represent incipient amyloidosis, they commented that clinically the lesions were easily removed and did not recur, and hence differed prognostically from those considered to be definitely amyloid. The pathologic features that they considered characteristic of true amyloid lesions were: (1) amorphous amyloid material occurred mainly in the form of "flakes" or "concentric layered masses" and (2) this amyloid material reacted characteristically with one or more of the so-called amyloid stains. They found crystal violet stain the most satisfactory for the identification of amyloid material.

In their pathologic study of five cases, Kreissl, Muirhead and Darrough[3] emphasized that in all these larynges, fibrinoid changes, as noted by Huebschmann, were prominent. At times, the smudgy, eosinophilic appearance of the connective tissue suggested the early phases of amyloid deposition; hence it appeared that collagen degeneration constituted an integral part of the local process. Microscopic hemorrhages seemed partially related to similar disintegration of the walls of the small blood vessels of the area. These authors commented that "apparently primary amyloid deposition may follow a pattern not unlike that of other mesenchymal disease. A local form and a systemic or disseminated form are observed, the former being more prevalent. The true relationship betwen these forms is not established. Fibrinoid degeneration of the adjoining tissue at times constitutes a part of the local process. This may not be significant but should be further studied."

TREATMENT

In the treatment of primary amyloidosis of the larynx, a conservative and expectant course is usually advised. Unless surgical removal promises to relieve the hoarseness which is usually the only complaint, operation, as a rule, is not recommended. If dyspnea supervenes, or the tumor shows a tendency to progression and recurrence, as in the case reported by Clerf,[11] then thyrotomy or some other procedure may be necessary. Patients in whom an amyloid tumor of the larynx is diagnosed are advised to return for follow-up examinations at frequent intervals, so that any progression of the

lesion may be dealt with. Generally, the lesion develops slowly and in many cases is self-limiting.[9]

Numerous medical treatments have been tried in the past, more particularly in the cases of secondary amyloidosis, and of these, liver extract has appeared to be the most promising.[12]

REPORT OF CASES

Before presenting a brief resumé of our own cases, the following record, furnished by Dr. Irl Blaisdell, of Syracuse, of a case of laryngeal amyloidosis treated by ACTH, is reported.

A man, aged 63, was first examined on June 2, 1952. He was complaining of hoarseness which had been present for the past year and had become progressively worse during the last two months. Examination of the nose, sinuses, ears and nasopharynx revealed no significant abnormalities. A mirror examination of the larynx showed an irregularity and fulness of the left cord, particularly in the posterior half. There appeared to be a small mass on the inferior surface of the posterior portion of the cord. On June 11, a direct laryngoscopy was performed under local anesthesia, and this revealed an indurated lesion, extending from the anterior commissure back to the arytenoid and beneath the surface of the left cord. Movement of the vocal cord was definitely limited. The lesion was round and smooth. Tissue was removed from the anterior and posterior portions of the lesion; histologic examination of these showed masses of homogeneous material with a foreign-body giant-cell reaction. Special stains confirmed the presumptive diagnosis of amyloidosis.

The patient was sent back to the referring physician who was advised to administer ACTH for two weeks. This treatment was carried out in the hospital in the patient's home town. He received a dosage of 100 milligrams of ACTH per day, and had no adverse reaction until the fourteenth day, at which time he became somewhat disoriented, and the medication was stopped. During this two-week period, his voice improved and his physician reported that the mass in the larynx seemed to disappear.

Re-examination by Dr. Blaisdell two months later, on August 15, showed that there was no limitation of motion of the left vocal cord, and the mass had definitely decreased in size. Another follow-up examination, on September 2, gave evidence of continued im-

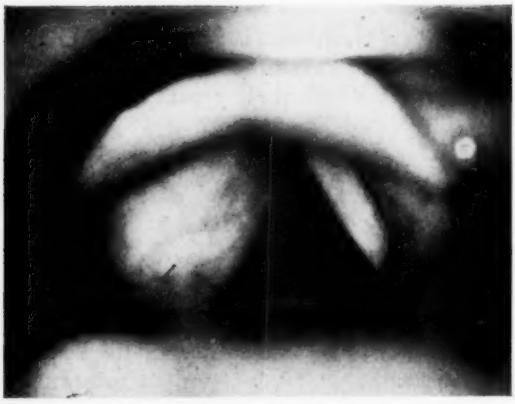

Fig. 3—Photograph of the larynx (Case 1) taken from a colored motion picture.

provement. The patient's voice was quite clear and he himself said that he could speak better than for some time past. A small quantity of abnormal tissue was still present beneath the left cord posteriorly, but the anterior portion of the mass had completely disappeared. It was Dr. Blaisdell's opinion that ACTH had caused definite improvement, and a second course of treatment for this patient was being planned.

Case 1.—A married woman, aged 33, had a mild huskiness of the voice for six months before she was first examined, in April, 1951. A laryngoscopy was performed on April 6, 1951, and the biopsy was positive for amyloid. The mass filled the right ventricle, overhanging the glottis and extending over the right false cord (Fig. 3). The mucous membrane over the amyloid mass was not ulcerated. This lack of ulceration is of diagnostic significance. By direct laryngoscopy, when the amyloid mass was displaced laterally, a normal true cord with normal movements was revealed. For this reason, voice impairment in this patient was slight. Physical examination showed no evidence of infection or amyloidosis elsewhere in the body.

In July, she was readmitted to the hospital for a series of cortisone injections directly into the tumor mass. The injections, each

Fig. 4.—Photograph of the larynx (Case 2) taken from a colored motion picture.

containing 25 milligrams of cortisone in 1 milliliter of saline solution, were made through a laryngoscope, using a long laryngeal needle and a tuberculin syringe. A series of eight injections daily were administered; during the period of treatment, there was no appreciable change in the size of the mass, nor was there any improvement in her voice. The cortisone used in this manner caused no toxic signs nor evidence of systemic effect.

Another course of four local injections of cortisone was given six weeks later, and again this treatment had no noticeable effect on the size of the tumor. Later, cortisone was administered by mouth, 200 milligrams daily for eight days and then 100 milligrams for six days. On the next day, the patient exhibited a severe reaction, with swelling of the face and of the ankles and pronounced vertigo, so the medication was discontinued.

Follow-up examinations were made in June, 1952, and again in February, 1953. The mass had not decreased in size, and probably eventually will require thyrotomy for its removal.

CASE 2.—A woman, aged 32, consulted us early in 1951, with a complaint of severe hoarseness, which had existed for two years. A mirror examination revealed several small red masses on the edge

of both vocal cords anteriorly and a larger reddish mass under the anterior commissure (Fig. 4). This patient had severe voice impairment. Direct laryngoscopy was done, and microscopic examination of multiple specimens of tissue showed the presence of amyloid deposits. A thorough physical examination was made to rule out amyloidosis in other organs and systemic disease.

This patient received eight injections of 25 milligrams of cortisone directly into the amyloid masses in July, 1951, and four injections of cortisone in September, 1951.

Examination in December, 1951, showed that the masses were smaller, particularly the one under the anterior commissure, and that the voice had improved. She did not return for observation for a year, and came in then only at our request. She stated that her voice had improved so much that she felt there was no need for further treatment.

The last examination of this patient, in February, 1953, showed that the improvement in laryngeal appearance and in the voice had been maintained.

COMMENT

The administration of cortisone in these cases was undertaken on an empirical basis, but a review of some of the recent literature on experimental amyloidosis has revealed a most interesting study by Gunnar Teilum,[13] of Copenhagen, reported in 1952, which suggests a rationale for the use of cortisone in primary amyloidosis and a possible explanation for the improvement noted in Dr. Blaisdell's case and in our second case in which the amyloid deposit was of the diffuse type.

After reviewing a considerable number of recent experimental studies,[14-26] it seemed obvious that Teilum's work has correlated the various experimental, pathological and clinical findings, with appraisal of their significance in such a way as to answer certain questions about amyloidosis more satisfactorily than has been possible heretofore. His observations and conclusions offer much elucidation of the subject, of interest to clinicians.

Teilum stresses that the mechanism whereby the hormone of the adrenal cortex maintains the structure and function of mesenchymal tissue in general is an indirect one, mediated by ascorbic acid. Well-

defined structural and functional phases in the natural diseases and in experimental disorders of the mesenchyma, which may in some way be associated with a disturbance of some enzyme system, such as ribonuclease, hyaluronidase, seem to be controlled by an interaction of a hormone of the adrenal cortex of cortisone type with ascorbic acid. These findings offer an approach to the elucidation of the following: (1) the pathogenesis of amyloidosis, (2) certain apparently antagonistic effects of cortisone and ascorbic acid on the mesenchymal tissue, and (3) the mechanism of action of cortisone and ascorbic acid on mesenchymal tissue in general.

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XXXIII

THE LAMINAGRAM AS AN AID IN THE DIAGNOSIS OF DISEASES OF THE LARYNX

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In the evaluation of laryngeal disease, the laryngologist should employ all appropriate methods of investigation to aid in the correct diagnosis. Not only should there be a thorough history, a careful clinical study by indirect and direct laryngoscopy, and laboratory examination of biopsy material, but there should also be a competent radiographic study of the diseased larynx.

The larynx has for many years been visualized by means of a lateral, soft-tissue radiograph of the neck. Credit for the initial use of this projection goes to Coutard.[1] Only by means of the lateral, soft-tissue view can the epiglottis, tumors of the anterior and of the posterior commissure, the post-cricoid area, and of the arytenoid area be best seen. The use of barium to outline the related pharyngeal structures need be resorted to only occasionally.

More recently laminagraphy has been included as a method of investigating laryngeal disease. By means of this radiological procedure, a roentgenogram is produced having only one section or plane of the body in sharp focus. This plane is approximately 0.5 to 1 cm in thickness while planes lying above and below are blurred and obliterated. This blurring is the result of motion in opposite directions of the focal point above the patient and the film below the patient during exposure. A line drawn from the focal point to a fixed point

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Fig. 1.—The first laminagram to appear in the literature; published by Dr. Felix E. Leborgne in the "Revista de Tuberculosis del Uruguay" Tomo V No. 4, 1936. Reproduced by permission.

on the film would rotate, during their motion, through an axis lying in the plane of focus. As a result, the shadow cast on the film by a point at the level of the axis of rotation will be in the same position throughout the excursion of the tube and the film, whereas the shadow cast by a point either above or below the axis will strike the film at a different position throughout the excursion resulting in a linear blur. This blurring will obviously be greater the farther the point is from the axis. Sections at various levels in the body can readily be obtained merely by changing the level of the axis of rotation. This procedure was first suggested by Bocage[2] in 1921; and was first applied by Vallebona[3] in 1930 to a study of the skull. Actually, Vallebona's method involved rotation of the patient on an axis related to a stationary film and a focal point, while Bocage's systems are the basis of present-day laminagraphy. The mathematical explanations of his methods have been worked out by Ziedses des Plantes[4] and by Grossmann[5] in 1935. An excellent historical review and mathematical analysis is given by Andrews[6] and by Andrews and Stava.[7]

Fig. 2.—Position of the patient.

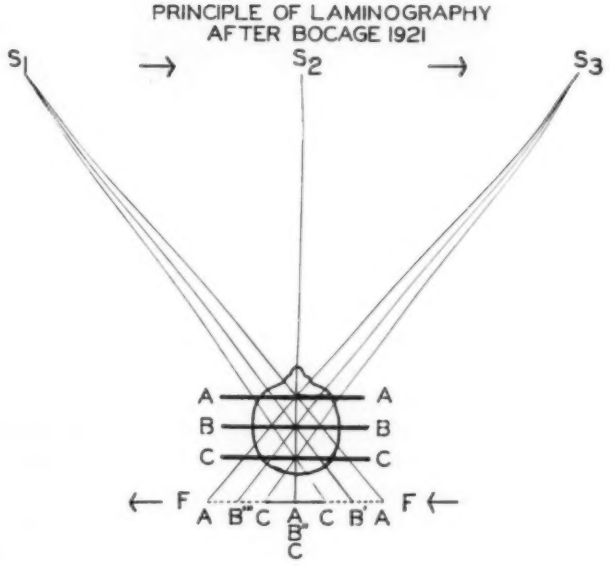

Fig. 3.—S1 to S3, excursion of the focal point; F to F, excursion of the film; Plane B, Plane of the laminagram; Plane A and C, Blurred.

Fig. 4.—Laminagram of the normal larynx.

Laminagraphy as applied to the study of the larynx should be credited to Felix E. Leborgne[8, 9] of Montevideo. In 1947 it was the senior author's privilege to visit Leborgne in Montevideo and to become familiar with his technique and to observe the value of the laminagram in diseases of the larynx.

In practice the larynx presents an almost ideal structure for laminagraphy. The air outlining the soft tissue of the respiratory passages forms an excellent contrast medium permitting easy visualization of the true and false cords, the ventricles, the trachea and immediate subglottic area, the pyriform sinus, and the vestibule. In addition, calcification of the laryngeal cartilages, which interferes with good visualization in the intrapharyngeal method actually improves the details in the laminagraph by clearly outlining the cartilages

in section. The permanent record provided by the laminagram for future comparison and the ability to compare the two sides on the film are of obvious value.

The apparatus employed by us consists of a conventional Bucky table converted with a motor-driven, detachable laminagraph apparatus which is available for almost any machine. The technique used is as follows:

Target to film	41.5 in.
Target excursion	14 in.
50 Milliamperes	
48-58 Kilovolts	
3 second exposure	
Intensifying screen with Potter-Bucky grid	

Positioning of the patient is done in such a way as to obtain comparable films in succeeding sections and in later studies. The patient lies supine with his neck halfway between full flexion and full extension. The head is fixed with sandbags and an adhesive plaster strap is placed under the chin and stuck to the sandbags for additional stability. The beam is then centered halfway between the laryngeal prominence and the tip of the chin. If the ventricles are seen in the previously taken soft-tissue lateral view of the neck, a second lateral view is taken with the patient positioned on the laminagraph table using a portable machine in order to get the ventricles in a vertical position and produce undistorted sections through the ventricle.

Laminagraphy has been used as a routine procedure at the Massachusetts Eye and Ear Infirmary only during the past year. In spite of the short time in which this additional method of examination has been used, our volume of cases has been such that, in our opinion, laminagraphy is of value:

(1) In confirming the findings of both indirect and direct laryngoscopy.

(2) In furnishing a permanent visual record of the lesion.

(3) In determining the exact limits of a subglottic extension.

(4) In visualization of the regression (or extension) of the disease during treatment by external irradiation.

(5) As an aid in those patients with short, thick necks and a backward tilt of the epiglottis who are difficult to examine under local or general anesthesia.

CONCLUSIONS

A thorough history, a careful indirect and direct laryngoscopy and a microscopic examination of biopsy material removed from the lesion are procedures essential in the investigation of laryngeal disease.

Laminagraphy is an additional method of examination that will aid the laryngologist in the care of the patient with lesions involving the larynx.

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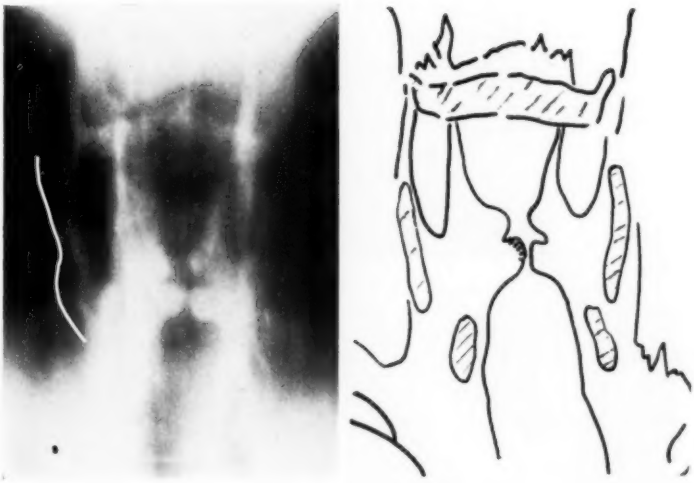
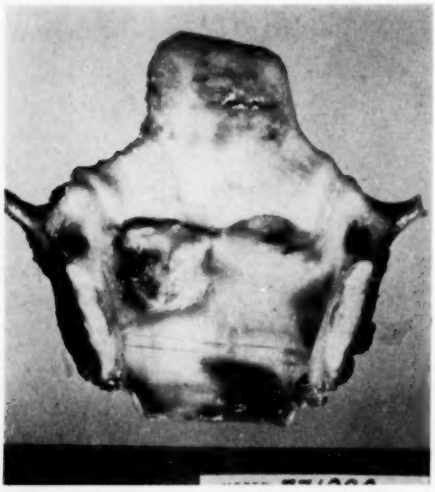
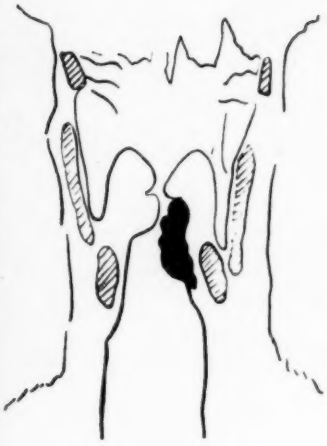
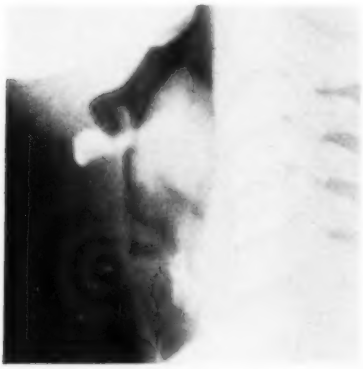


Case No. 700073. Male. Aged 71. Hoarse for two months. Laryngoscopy—The right cord thickened and red. No limitation of motion. Biopsy—Chronic inflammation. Laminagram—Normal left true cord. Ventricle well visualized. Right cord swollen with narrowing of the vestibule.

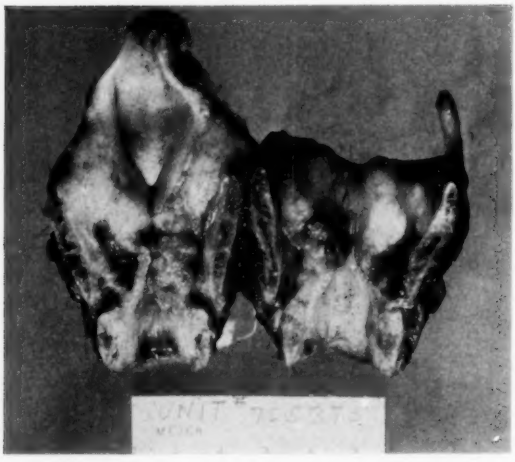




Case No. 771090. Male. Aged 39. Hoarse for one year. Laryngoscopy—new growth involving entire left true cord extending subglottically. Biopsy—Epidermoid Carcinoma Grade 2. Laminagram—Excessive new growth of left vocal cord with subglottic extension. The ventricle is obliterated. Treatment—Total laryngectomy.

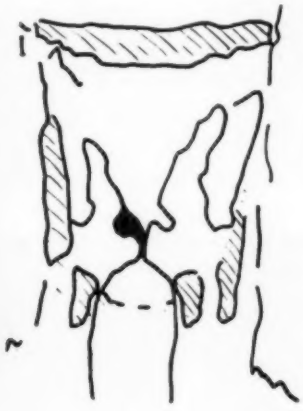


Case No. 765508. Female. Aged 29. Sensation of "lump" in the throat. Laryngoscopy—Large, smooth mass filling the hypopharynx and obliterating a view of the vocal cords. Lateral radiogram shows the mass to be supra-glottic extending above the tip of the epiglottis. Biopsy—Lymphosarcoma. Laminagram—revealed mass coming from the right pyriform sinus and entirely supraglottic.

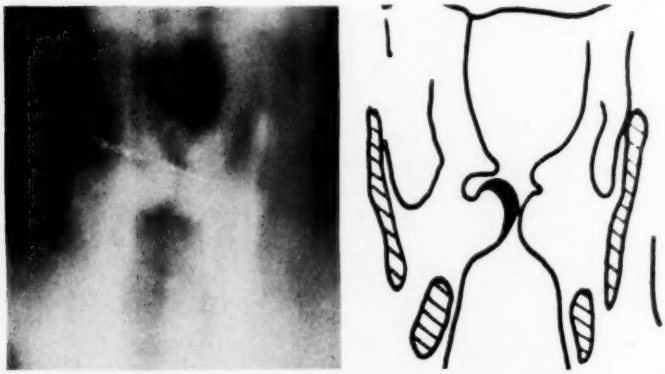


Case No. 765375. Male. Aged 72. Hoarse for five months. Laryngoscopy—Large mass involving entire right vocal cord with fixation. Biopsy—Epidermoid Carcinoma grade 3. Laminagram—The true cord was replaced by shaggy mass filling ventricle and pushing the false cord upward, with subglottic extension. Treatment—Total laryngectomy.

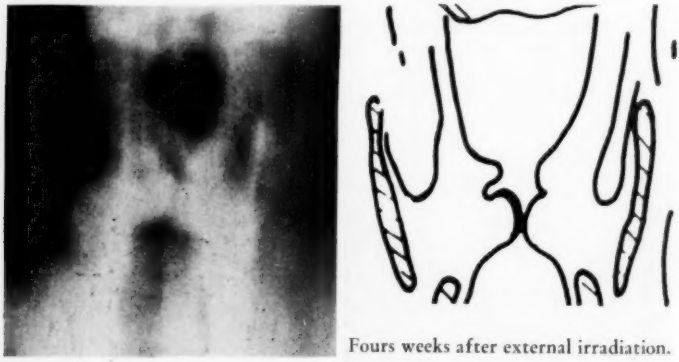
Case No. 806320. Male. Aged 60. Trouble with voice since 1947. Two biopsies in 1947 showed chronic inflammation. In 1953 was hoarse for two months. Laryngoscopy—localized lesion of the midportion of the right cord. Biopsy—Carcinoma grade 2. Laminagram—shows the lesion of the right cord. Treatment—Laryngofissure.



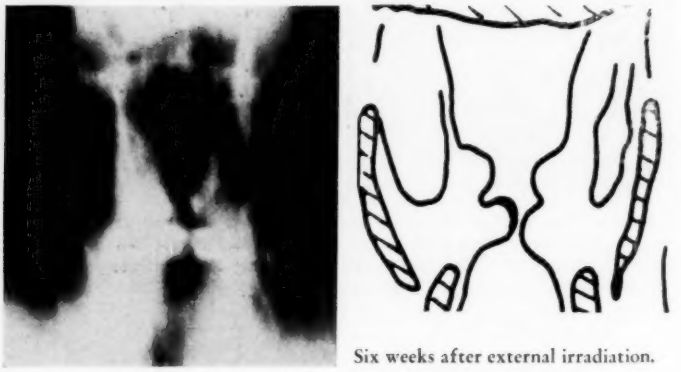
Case No. 801705. Male. Aged 60. Hoarse over one year. Laryngoscopy—new growth on midportion of right vocal cord. No limitation of motion. Biopsy—Epidermoid carcinoma grade 1. Laminagram—demonstrates lesion. Treatment—Laryngofissure.



Case No. 766284. Male. Aged 45. Hoarse for three months. Laryngoscopy—thickened left vocal cord. Biopsy—Carcinoma grade 2.

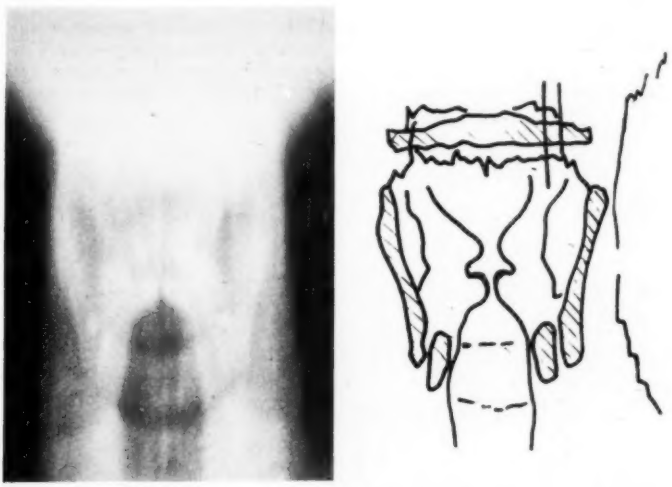


Fours weeks after external irradiation.

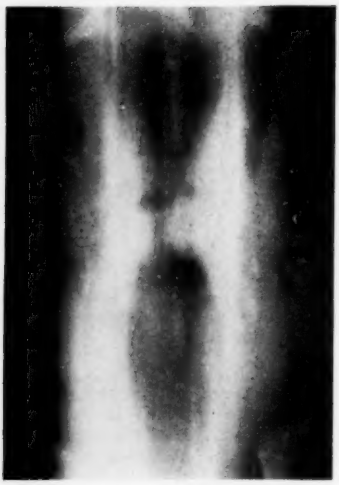


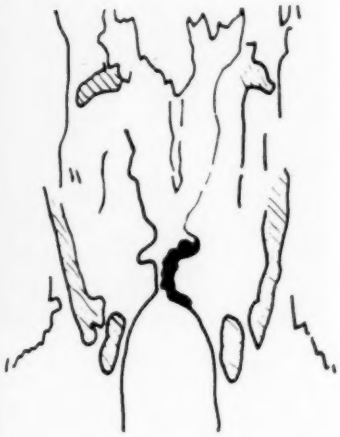
Six weeks after external irradiation.

Case No. 769992. Male. Aged 42. Hoarse off and on for one year, constantly for four months. Laryngoscopy—new growth involving the upper surface in its midportion of the right cord. Laminagram confirms the direct findings. Biopsy—Carcinoma grade 2. Treatment—Laryngofissure.

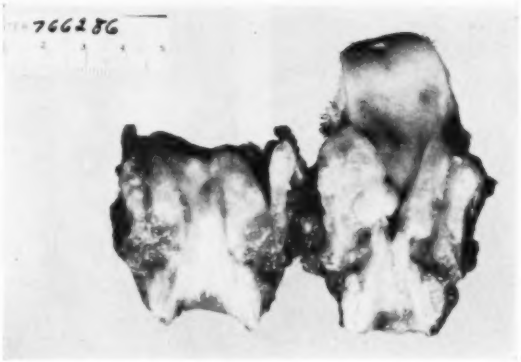


Case M. W. Female. Aged 36. Many complaints of her throat some of which suggested the possibility of a laryngocele. Laryngeal examination negative which was confirmed by the laminagram.



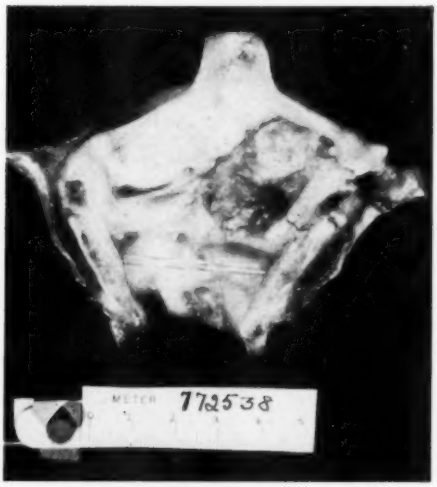


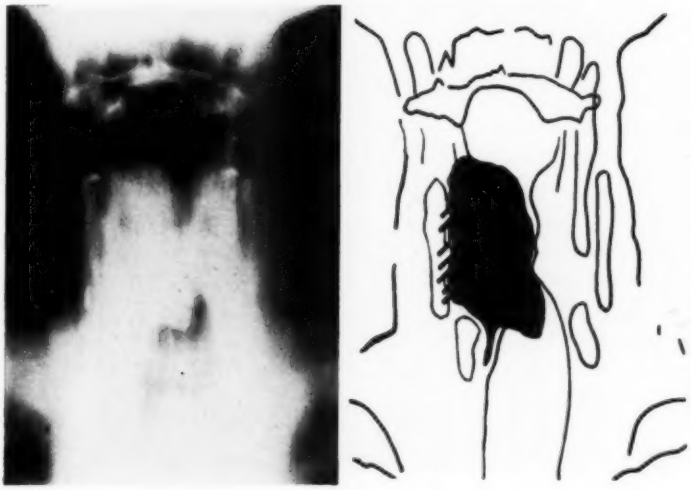
Case No. 799630. Male. Aged 71. Intermittent hoarseness three years. Constant hoarseness for six months. Laryngoscopy—Entire left vocal cord involved with fixation. Biopsy—Epidermoid carcinoma grade 3. Laminagram—demonstrates the lesion. Treatment—Total laryngectomy.



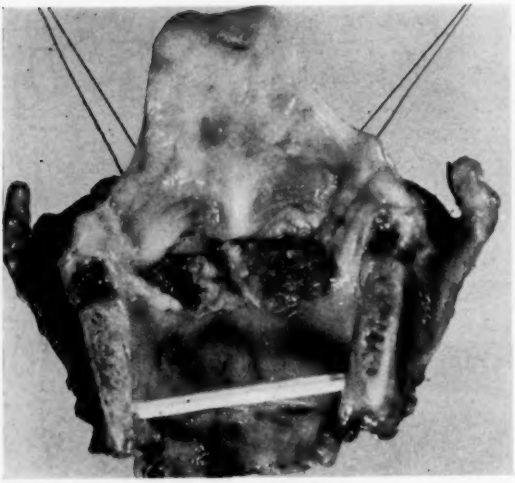
Case No. 766286. Male. Aged 74. Alteration in voice one year. Hoarse-constant three months. Large fungating lesion left false cord and right true cord. Biopsy—Epidermoid Carcinoma grade 3. The laminagram shows enlargement of the left false cord with obliteration of the left ventricle and extension to the ary-epiglottic fold.

Case No. 771594. Male. Aged 56. Hoarse for three weeks with pain upon swallowing. Laryngoscopy—proliferating new growth at base of epiglottis extending to ary-epiglottic fold and right supraglottic area of larynx. Biopsy—Epidermoid Carcinoma Grade 2. Laminagram shows the extent of the lesion. Treatment— Total laryngectomy.

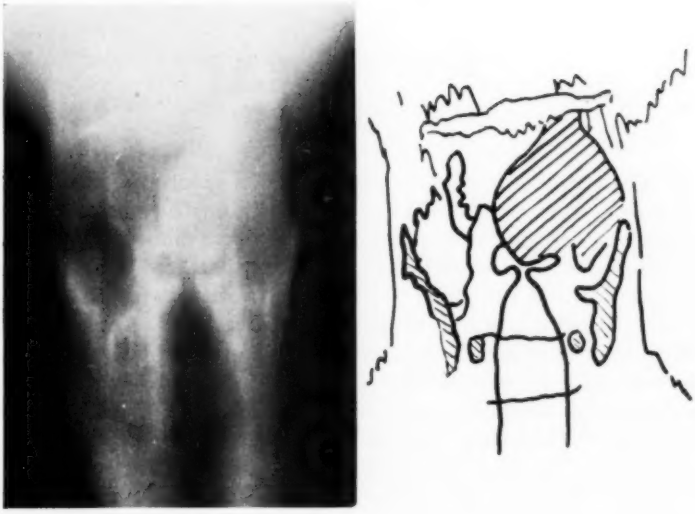




Case No. 722538. Male. Aged 70. Hoarse for three years. Pain referred to right ear. Laryngoscopy—Lesion of the right vocal cord involving ventricle, false cord, and extending almost to the ary-epiglottic fold. Biopsy—Epidermoid Carcinoma Grade 3. Laminagram—Extension of lesion of the right side of the larynx, obliteration of the ventricle, and extension upward toward the ary-epiglottic fold. Treatment—Total laryngectomy.



Case No. 766161. Male. Aged 45. Intermittently hoarse for two or three years—continuous for three months. Laryngoscopy—fungating lesion of both vocal cords with subglottic extension and fixation of the right cord. Laminagram—marked enlargement of both true cords with obliteration of the ventricles and subglottic extension on the right side. Biopsy report—Epidermoid carcinoma grade 1. Treatment—Total laryngectomy.



Case No. 212878. Female. Aged 36. This patient has been under observation since 1936 for an angioma involving the left false cord. The laminagram shows the lesion occupying the space of the left false cord and extending upward to the ary-epiglottic fold and piriform sinus. The ventricle is closed due to pressure from above. The true cords are not involved.

XXXIV

THE CLINICAL SIGNIFICANCE OF KERATOSIS OF THE LARYNX AS A PREMALIGNANT LESION

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The detection of a premalignant lesion before the development of clinical evidence of invasion presents a challenge to physicians. Frequently the challenge is not accepted, thus allowing for the development of an attitude of complacency, both on the part of the physician and of the patient, lulling them both into a feeling of false security based entirely on the negative character of a single histologic study. In keratosis of the larynx that is histologically benign one cannot speak of absolute benignity for the factors responsible for malignant degeneration may be activated to exert an uninhibited stimulus. When these changes may occur cannot be predicted, so that benign keratosis must always be considered as fertile soil for malignant development, making subsequent, frequent and prolonged periods of observation and histologic study essential.

The relationship between keratosis and carcinoma is controversial. In the presence of laryngeal keratosis carcinoma can easily be missed because of its limited extent at its inception. Are laryngeal keratosis, then, which are not malignant when first seen to be regarded as precarcinomatous? If so, is it in the sense that such degeneration is usual, occasional, or rare? It is our feeling that keratosis of the larynx is a premalignant lesion in a certain definite percentage of cases, and when the disease persists carcinoma is the usual outcome.

In a series of 125 patients with keratosis of the larynx studied at Jefferson Hospital (Table 1) the youngest was 32 years and the oldest 82 years with an average age of 54. The duration of symp-

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TABLE 1.

KERATOSIS OF LARYNX IN 125 PATIENTS

Age	32 to 82 years	
Sex	Male	117
	Female	8
Duration of symptoms	2 weeks to 10 years	
Location of lesion	Unilateral	89
	Bilateral	36

toms ranged from two weeks to 10 years, the average being one year. There were 117 males and eight females. The keratosis was unilateral in 89 cases and bilateral in 36 cases.

The clinical term keratosis does not describe a definite and specific disease. While keratosis may be simply a surface manifestation of an underlying disease process it may also represent the sole pathological lesion. Keratosis implies a whitish patch, either flat or raised, smooth or nodular, localized or diffuse, surrounded by normal tissue or by an area of inflammation. The clinical picture is so variable that it necessitates confirmation by an exact histologic study to be of value. Histologically the findings consist of incomplete desquamation and abnormal cornification of the epithelial layer of mucosa with the formation of excessive keratin on its surface. In places the epithelium may dip deeply into the connective tissue, but the basement membrane remains intact. Subepithelial inflammatory cells are present and when these are numerous some observers have felt that they indicate a predilection for carcinoma. In keratoses of long standing, fibrous tissue develops in the subepithelial layers.

The cause of laryngeal keratosis has generally been attributed to chronic irritation of long duration. Metaplasias of the mucous membranes of other parts of the body have been frequently observed, and in about 50% of the cases studied by Niskanen[6] metaplasias were found in the bronchi in association with various inflammatory diseases of the lung. It would appear that keratosis of the larynx is equally common

following chronic irritation. The exact mechanism of these chronic, irritating factors producing epithelial metaplasia has not yet been fully uncovered. The irritation from smoking has long been considered a salient factor. In this series only 11.2% were non-smokers. This figure may be significant when compared with the smoking habits in the cross-section of the normal population as determined in Columbus, Ohio, in which 34% were non-smokers.[4] The majority of the smokers admitted to excessive use of tobacco. Yet in certain individuals who have submitted their larynges to this form of irritation over many years no such lesion has developed. Factors other than chronic irritation are probably necessary to produce this change. In some individuals this heaping up of epithelium may represent a regenerative process in an attempt to overcome the damage from the chronic inflammation. When the damage is repaired and the causative factor removed, epithelization becomes complete and proliferation ceases. The danger lies in the fact that the reaction to the stimulation may at some stage overstep its normal bounds and initiate a premalignant phase, eventually to be followed by malignant invasiveness.

In some instances keratosis has completely disappeared without any form of treatment. The keratosis experimentally produced by avitaminosis and deficiency in estrogenic hormonal substances is reversible. This reversability may extend to those cases in which chronic irritation is a factor. Clerf[1] pointed out that it is difficult if not impossible to predict the outcome in any one case.

In carcinoma of the larynx of low-grade malignancy, before there is fixation and invasion, keratosis may be the first sign because of the well-differentiated epithelium and formation of keratin. Infiltrating carcinoma may begin from a simple keratotic change, or the keratosis may appear as a stage in the malignancy, or develop on the surface of the carcinoma after it has formed.

The cases forming the basis of this report were classified clinically into three groups: (1) those which were carcinoma when first seen (57 cases), (2) those which were first benign and later developed into carcinoma (27 cases), and (3) those which never showed evidence of malignant degeneration, some after years of observation (41 cases).

It is the second and most interesting group of 27 patients first diagnosed as keratosis and progressing to carcinoma with which this discussion is particularly concerned. There were six cases in which the malignancy occurred less than six months after the first biopsy.

TABLE 2.

GROUP OF 27 CASES OF KERATOSIS OF THE LARYNX WHICH SUBSEQUENTLY BECAME MALIGNANT INDICATING TIME INTERVAL BETWEEN THE BENIGN AND MALIGNANT PHASES.

TIME	NUMBER
under 6 months	6
½ to 1 year	4
1 to 2 years	6
2 to 3 years	3
3 to 4 years	2
4 to 5 years	2
over 5 years	4

These cases were undoubtedly malignant when first examined, but the carcinomatous change was not present in the biopsy specimen at that time. Subsequent histologic study revealed the malignancy which must have been present earlier but was not represented in the initial tissue. Of particular significance is the occurrence of carcinoma in four patients beyond a five-year period. In three of these there were intervals of from eight to ten years between the keratosis and the carcinoma (Table 2).

In observing keratosis of the larynx clinically we have felt that those lesions which exhibited a definite inflammatory zone around the periphery of the keratosis were more apt to be malignant or undergo malignant degeneration than those in which there was no inflammatory zone. In addition, those lesions which were irregular in their projection above the surface with a papillary appearance more often were found to contain carcinoma than those in which the lesion was flat with no sharp line of demarcation between the keratosis and the normal mucosa.

In those cases which undergo malignant degeneration the first change commonly observed is an alteration in the intraepithelial

Fig. 1 (A).—Histologic section of keratosis of vocal cord. Area of mucosal change was regarded as premalignant and not definite enough to be diagnosed as intraepithelial carcinoma.

cellular structure which is now designated as carcinoma in situ. This stage of malignant cellular change limited to the epithelium, and without invasion of the basement membrane, may last for many years, and its exact potentialities are in doubt. On the other hand this change may be the first indication of invasive carcinoma. At times one may find carcinoma in situ adjacent to invasive carcinoma or invasion may be present in other parts of the lesion. The distinction between intraepithelial carcinoma and simple keratinization, while difficult, is of prime importance.

Formerly the pathologic diagnosis, as benign or malignant, was used to determine treatment to the exclusion of all other factors. The uncertainties of the now existent borderline conditions of premalignant and preinvasive carcinoma has rendered our selection of treatment debatable.

Repeated histologic examination of different portions of the keratotic lesion may be needed to make an accurate diagnosis. The

management of the case depends upon the clinician's evaluation of the lesion. He cannot accept a negative pathologic report as unequivocal and advise treatment solely on the basis of this finding. The clinical appearance of the lesion and the laryngologist's judgment are equally potent factors in determining the type of treatment.

With the modern emphasis on prevention and early diagnosis of carcinoma we may be carried away with the desire to prevent or eradicate the disease without properly verifying the diagnosis. This is especially true in dealing with carcinoma in situ, and care must be taken not to make an erroneous diagnosis on a few suspicious cells. Vast pathologic experience is needed to make this diagnosis, and there is room for error even by the most experienced pathologist. When this latter lesion of carcinoma in situ is present it is our feeling that conservative management can be followed. When the lesion is not extensive and is limited to a small portion of the vocal cord, it can be excised surgically. In its removal, however, we believe that the perichondrium should not be included so that a better functioning vocal cord is formed. Simple excision of the keratosis with its carcinoma in situ should be sufficient. In 19 cases of this group the entire lesion was removed in the biopsy specimen, and when thyrofissure was performed no residual carcinoma was found histologically in the excised tissue. This type of early carcinoma can also be treated with irradiation therapy, which has been reported as curative in a high percentage of cases. Such a lesion would also appear ideal for electrosurgical removal with suspension laryngoscopy as advocated by New and his associates[5] and Le Jeune.[3]

In addition to the 41 cases of initial benign keratosis there were nine patients who developed it following laryngofissure for keratotic carcinoma. Two of these occurred at the operative site, five developed in other portions of the larynx unconnected with the original lesion, and two spread throughout the larynx. Whereas this subsequent formation of keratosis might be independent of the former lesion it suggests that there is a predilection for keratosis in certain individuals, and when removed in one location it may occur in other areas of the larynx. The following case illustrates the tendency to recurrence:

CASE 1.—In 1948 a 37 year old white male was examined for hoarseness of three months' duration. He smoked at least three packs of cigarettes daily. On indirect laryngoscopy a raised, whitish area was seen on the left vocal cord

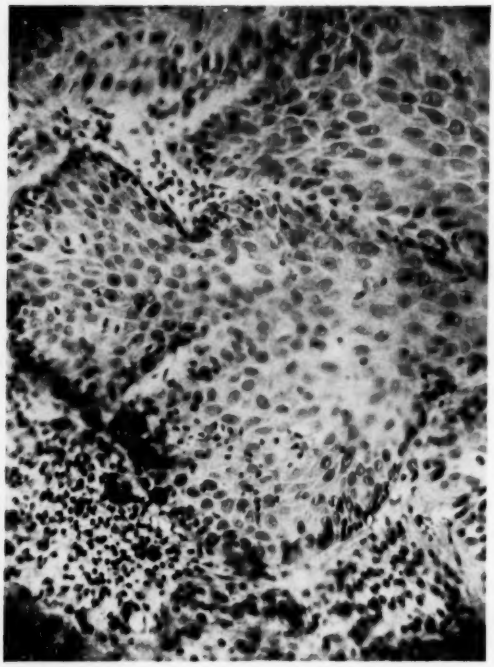

Fig. 1 (B).—Higher magnification of suspicious area of mucosal malignant change.

which proved on histologic examination to be squamous cell carcinoma. The lesion was treated by laryngofissure with removal of the left vocal At this time there was no abnormality of the right vocal cord. One year later benign keratosis developed on the left vocal band at the operative site. In 1950 the right vocal cord which had previously been normal became the site of benign keratosis, and in 1951 squamous cell carcinoma developed in the right vocal cord. Since the entire lesion on the right was removed by biopsy no further surgical treatment was carried out at that time. The patient resumed smoking, and two years later, in 1953, carcinomatous changes limited to the mucosa of both vocal bands returned. Irradiation therapy was administered with disappearance of the lesion, and close watch is being maintained on the condition of the larynx.

In 1942 Graham[2] stated that most cases of keratosis can be handled with ease by removal of the lesion. While it is true that some cases are managed by this method, it has been our experience that in

the majority of cases successful treatment has proved more difficult. A benign keratotic lesion with no histologic evidence of malignancy can be removed, and the elimination of all forms of irritation may help prevent its return. When the lesion recurs after this treatment the problem becomes more complicated. The following case is an example of keratosis which remained benign but recurred over a six year period:

CASE 2.—Hoarseness was present for one month in a 52-year-old white male, who had smoked a pack of cigarettes daily for many years. The entire right vocal cord was involved by a raised, whitish nodular lesion on the edge and upper surface.

Forceps removal of the material at direct laryngoscopy was done, and the histologic diagnosis was keratosis. The lesion returned the following year, and the pathologic report was still keratosis but with some irregularity suspicious of carcinoma. For the next five years the lesion would disappear and recur, and histologic study at yearly intervals consistently revealed benign keratosis. Shortly before his death from coronary thrombosis keratotic material was removed from the right vocal cord, and no evidence of malignancy could be demonstrated.

When the keratosis is benign but is more extensive and involves an entire vocal cord or extends over a considerable portion of both vocal cords the problem of management becomes even more involved. If the keratosis persists after the elimination of all forms of irritation, is there anything else that can be done to eliminate the keratosis and prevent the carcinoma which will subsequently develop in many cases? Irradiation therapy was given to some of these patients, even though these well-differentiated lesions do not lend themselves well to this type of therapy. Radiologists have reported cases of keratosis with malignant degeneration, particularly in the buccal mucosa, in which irradiation therapy eradicated the carcinoma, but the keratosis persisted. The need for absolute safety against extension of the lesion is essential, yet the necessity for avoiding needless interference with the voice is extremely important. Surgical removal in this type of case would entail either a complete laryngectomy or else extensive intralaryngeal surgery, so that the voice would be markedly impaired. It is our feeling that irradiation therapy is beneficial in the treatment of extensive, persistent keratosis. This is illustrated in the two following case reports:

CASE 3.—In 1941 a 48-year-old white male complained of hoarseness of six months' duration. He admitted to smoking two packs of cigarettes daily. Mirror laryngoscopy revealed a raised whitish lesion on the anterior halves of both vocal cords. On the left side the involved cord appeared irregular surrounded by an

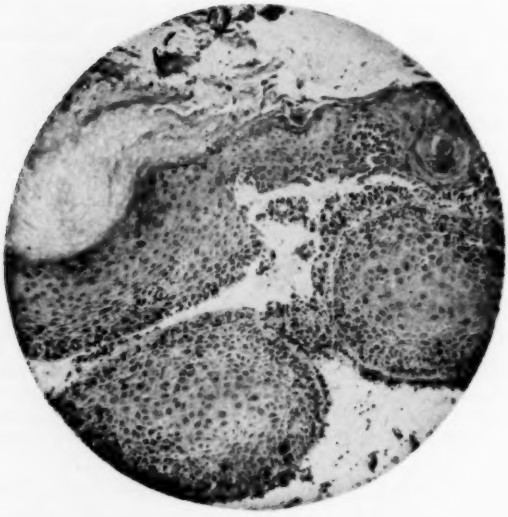

Fig. 2.—Eight years after the beginning of keratosis with premalignant characteristics invasive carcinoma was present on histologic section.

inflammatory zone. All of the material was removed with forceps at direct laryngoscopy and the histologic report was keratosis, suspicious of malignancy. The lesion recurred persistently and tissue examinations were repeated each year with pathologic reports varying from simple keratinization to keratosis, suspicious of malignancy (Fig. 1). In 1949, eight years after the original keratosis, the pathologist diagnosed squamous cell carcinoma of the left vocal cord (Fig. 2). Because of the generalized extent of the keratosis on both vocal cords the patient was treated with irradiation. He received a tissue dose of 6080 r delivered in a 5.5 cm field to the midline of the larynx over a period of five weeks. When last examined three years later generalized redness with edema and thickening of the mucosa was apparent, but there was no recurrence of either the keratosis or the carcinoma.

Case 4.—A 38-year-old white male complained of hoarseness of three months' duration. On examination of the larynx an irregular, whitish lesion involving the anterior two thirds of both vocal cords extending to the anterior commissure was seen. The tissue was heaped up in the middle third of the right vocal cord. On histologic study of the tissue benign keratosis was found. Since the keratosis recurred after removal frequent tissue examinations were made, the report always being keratosis. Approximately one year after the original biopsy squamous-cell carcinoma developed in the right vocal cord. Laryngofissure was performed, and the anterior portions of both vocal cords including the anterior commissure were removed. The specimen revealed squamous-cell carcinoma in the right vocal cord

and benign keratosis in the left vocal cord. Six months following operation keratosis began to reappear on the left vocal cord. It increased during the next four years until it again involved both vocal cords and extended onto both ventricular bands, involving almost the entire intralaryngeal mucosa. Irradiation therapy was given in the dosage of 4560 r delivered in a 25 sq cm field to the midline of the larynx over a five-week period. Following irradiation the keratosis completely disappeared, and when last seen the larynx remained free from it but exhibited post-irradiation changes.

SUMMARY

There are cases of laryngeal keratosis which have nothing in common with carcinoma. In another group it leads to carcinoma, and in a third group keratosis develops as a sign of carcinoma. Since it is impossible to tell which area will remain keratosis and which will undergo carcinomatous change, the elimination of the entire keratotic area should be practiced if at all possible. In simple keratinization marked improvement will follow within two or three weeks after the removal of all forms of irritation and abuse of the larynx. If the lesion has not exhibited definite clinical evidence of healing within this length of time, it must be regarded with suspicion. Persistent or recurrent keratosis requires repeated histologic study including the subepithelial layer to detect the malignant change which is the usual outcome. Treatment with irradiation may produce disappearance of the keratotic lesion while it is still in the premalignant stage. In those cases in which the first manifestation of carcinoma is keratosis, the management follows that of laryngeal malignancy. Distinction between simple keratinization and carcinoma in situ in the larynx can now be recognized by the pathologist, but the lesion should be evaluated by the laryngologist and the method of treatment dictated by his clinical experience.

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XXXV

CARCINOMA-IN-SITU OF THE LARYNX

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Carcinoma-in-situ of the larynx is being recognized with increasing frequency in biopsy material from cases in which the clinical diagnosis of carcinoma was not apparent. Undoubtedly this is due in large measure to a growing appreciation and awareness of the concept of an intra-epithelial, non-invasive stage of carcinoma by both laryngologists and pathologists coupled with an increased use of the biopsy in superficial lesions of the larynx. The disclosure of the presence of carcinoma-in-situ raises in each case a series of questions as to the pathogenesis of this legion, the criteria for its identification, the invasive potential that might be anticipated, and the most appropriate treatment for it. With these problems in mind, a review of our experiences with thirteen cases is presented.

The term carcinoma-in-situ was used by Broders[1] in 1932 to describe an early pre-invasive stage of intra-epithelial carcinoma that is recognized by the profound cellular alteration in intact surface epithelium. The realization of the true, potentially malignant nature of this lesion was expressed by the earlier term of intra-epidermal carcinoma, and dates back at least to the observations of Bowen (1912). In the last two decades a voluminous literature has recorded the increasing recognition of this pathological state. Although much of the work has concerned the uterine cervix, the possibility of this condition appearing in any epithelium was recognized early, as the report by Mallory[2] in 1940 indicates. Votaw[3] reviewed the evolution of the acceptance of this entity and described five cases of carcinoma-in-situ of the larynx. Additional mention appears to be confined to the case reports of Brighton and Altman,[4] Clerf,[5] Snitman,[6] Brun-

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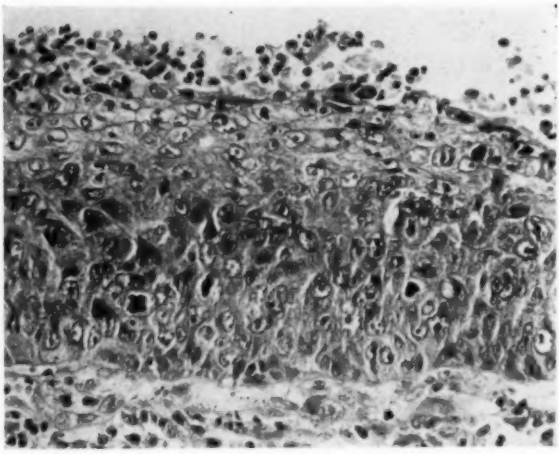

Fig. 1.—Carcinoma-in-situ, larynx. x 350. Mitotic figures prominent.

ner,[7] Gordon,[8] and the group of cases reported from the Presbyterian Hospital by Altman, Ginsberg and Stout.[9]

HISTOLOGICAL CRITERIA

The histologic features of carcinoma-in-situ are reasonably obvious, particularly in stratified squamous epithelium which is the site usually affected by the process. At first glance, the epithelium appears hyperchromatic and basophilic and the normal stratiform differentiation of the epithelium is lost (Fig. 1). The normal layers are indistinct or missing and the regular progression from basal cell layer to surface is no longer present. The basal cells vary in size and shape and their type is abnormally extended up through the more superficial layers, so that the normal single cell layer of basal units is no longer distinct. In the prickle-cell layer the intercellular bridges have usually disappeared. In this region the cells tend to be rounded or irregularly polyhedral; the nuclei vary in size and shape and large "bird's eye" nuclei with large nucleoli are present. The absence of a distinct and uniform basal cell layer and the occurrence of anisonucleosis throughout the thickness of the epithelium are the most arresting and convincing features to the observer. Mitotic

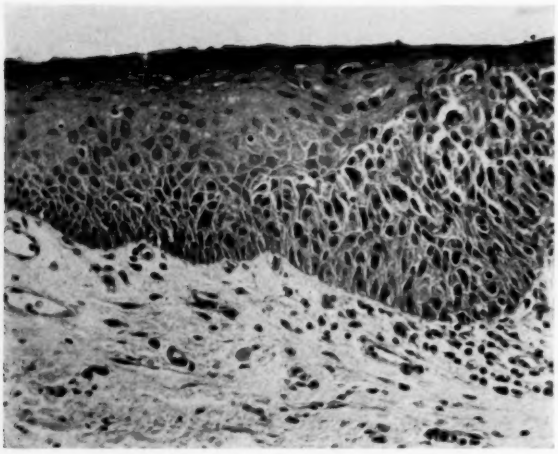

Fig. 2.—Margin of carcinoma-in-situ, larynx. x 350. The junction is diagonal.

figures are usually numerous and they can always be found in the area involved. These figures may be normal or atypic and muli-polar in appearance.

Irregular foci or keratinization can be found in any portion of the epithelial layer. In some cases this occurs near the surface, and occasionally the entire surface is covered by a keratotic or parakeratotic layer of considerable thickness. The appearance of the process of carcinoma-in-situ either without or with accompanying well-defined hyperkeratosis, suggests that this profound change can occur in previously normal epithelium or in epithelium which is the seat of keratosis.

Usually the layer of epithelium involved by carcinoma-in-situ is increased in thickness but this is not invariably so, as the same changes can be seen in extremely thin epithelial surface layers. The epithelium can extend in a flat unvarying layer or its epithelial pegs can be markedly deepened and widened. The supporting tissue varies from practically a normal state to one which is greatly changed by edema, congestion and inflammatory alteration. The presence of a

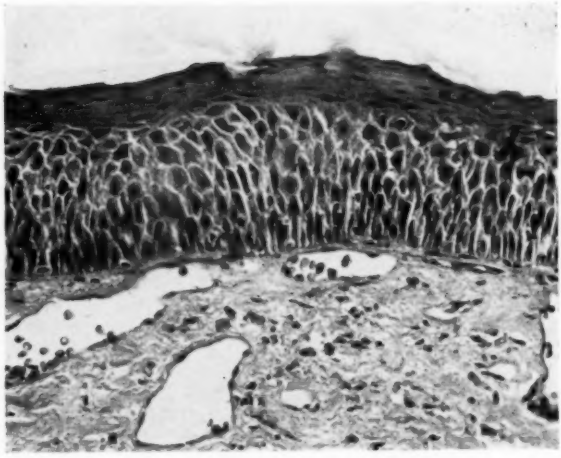

Fig. 3.—"Basal zone hyperplasia," larynx. x 350.

lymphocytic infiltration of considerable intensity just beneath the involved epithelium appears to suggest that invasion is imminent; it is seen most often when the large epithelial pegs intrude deeply into the supporting tissue.

The presence of in-situ carcinomatous change in epithelium is most obvious at the junction of unaltered and carcinomatous epithelium (Fig. 2). The junction line is usually sharp and distinct, except in those cases of carcinoma-in-situ developing in previous keratosis.

In approaching the microscopic diagnosis of carcinoma-in-situ, the difference between it and normal stratified squamous epithelium or simple keratosis is usually obvious, but it can be confused with a condition we refer to as "basal zone hyperplasia" (Fig. 3). In this latter lesion, the basal type of cell continues up through a portion of the prickle-cell layer developing a zone in which the cells are uniformly hyperchromatic and have a vertical polarity. Commonly this process terminates abruptly at the upper prickle-cell layer with a sharp zone of transition to horizontally oriented cells. Although the cells in this basal zone are hyperplastic and mitotic figures are

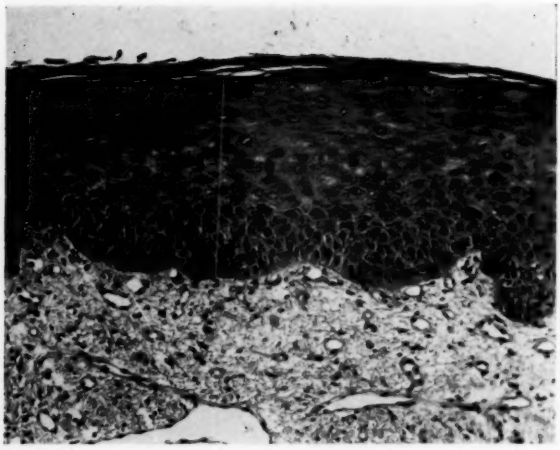

Fig. 4.—Keratosis, larynx. x 175.

encountered, the absence of anisonucleosis and the perfectly regular pattern presented by this change argue for the exclusion of this lesion from those identified as carcinoma-in-situ. We believe that this process or basal zone hyperplasia represents a transitional phase prior to carcinoma-in-situ in which the biological intent for malignant proliferation has not yet been declared. This process occurs as an isolated phenomena and also has been found to be associated with carcinoma-in-situ and invasive carcinoma.

The recognition of carcinoma-in-situ has developed more precise criteria for the non-malignant hyperplastic phenomena of the laryngeal mucosa. Keratosis is manifested by regular hyperplasia with a normal sequence of differentiation from the basal layer to surface, usually with an accentuation of the zone of keratinization (Fig. 4). Anisonucleosis is absent. Although mitotic figures can be anticipated, peculiar atypic mitoses and isolated bizarre giant cells are not encountered in simple keratosis. As in carcinoma-in-situ, the keratotic epithelium can vary markedly from a thin to a thick epithelial layer; usually it is greatly thickened, exceeding in width the average case of carcinoma-in-situ.

TABLE I.

NO.	CASE	AGE	SEX	DURATION OF HOARSENESS (MONTHS)	SMOKING	VOCAL ABUSE
1.	D. W.	37	M	3	Heavy	+
2.	P. B.	57	M	12	Heavy	0
3.	S. K.	44	F	9	No	+
4.	F. F.	29	M	1½	Moderate	0
5.	D. A.	34	F	1¼	Moderate	0
6.	T. T.	45	F	12	Moderate	0
7.	G. J.	68	M	Years	Heavy	0
8.	C. C.	78	M	5	Moderate	0
9.	F. B.	56	M	Years	No	+
10.	L. S.	50	M	15	Heavy	0
11.	A. S.	55	M	6	Heavy	0
12.	H. A.	40	M	12	Heavy	0
13.	H. B.	63	M	1½	Heavy	0

By definition, carcinoma-in-situ is confined to the surface epithelium and has not extended by invasion into the supporting connective tissue (Fig. 5). The process can continue down into the ducts of glands without violating this concept. There is always a clear-cut, regular and continuous demarcation between epithelium and connective tissue.

CLINICAL FEATURES OF CARCINOMA-IN-SITU

Since September of 1948 we have encountered 13 patients with superficial lesions of the larynx which on histopathologic examination revealed intra-epithelial carcinoma without invasive tumor (Table I). (The many cases of carcinoma-in-situ associated with invasive carcinoma are not being considered in this report.) The youngest patient was 29 years of age and the oldest 78. The average age was 46 and the median age was 48. Only three of the thirteen were women.

TABLE II.

CLINICAL APPEARANCE.

Leukoplakia	8	
Thick		5
Thin and punctate		3
Thickening	7	
Redness	8	
All larynx		1
Entire cord		4
Focal		3
Distribution		
One cord	11	
Entire		6
Partial		5
Both cords	2	

Hoarseness, the only symptom, was present in all patients and was continuous from one and one-half to fifteen months before diagnosis. Four patients had been intermittently hoarse for years. Of the entire thirteen, seven were heavy smokers; four smoked moderately, and two were non-smokers. One of the non-smokers, a woman, is one of the two patients who have had a recurrence. There probably had been vocal abuse in three patients. Two of these were salesmen and one was a lecturer.

Upon indirect or direct laryngoscopic examination there was no characteristic or distinctive picture. In fact, a clinical diagnosis of carcinoma-in-situ was not made in any patient prior to biopsy and the appearance in only two lesions suggested that they might be malignant.

(Table II) Leukoplakia was prominent in eight cases. In five of these the white coating was very thick and somewhat shaggy, and was the only visible lesion other than slight thickening of the area involved (Fig. 5). In three there was thin, punctate leukoplakia over slight thickening. The remaining five patients (Fig. 6) exhibited only redness and thickening. One entire cord was diffusely involved by a smooth, red swelling in three of these five cases, (Fig. 7) while the other two (Fig. 8) had localized irregular red thickening of only a part of a true cord. In all of the cases in this series the lesion was intrinsic in location. Both cords were involved in only two cases (Fig. 9). In seven cases all of one cord from anterior to posterior commissure was involved so that a laryngo-fissure operation could not be expected to safely remove all disease.

One larynx was especially interesting. The original lesion was a small, sesile, dark-red mass on the mesial border of the middle third of the right true cord. This lesion was stripped away and histopathological examination revealed carcinoma-in-situ with a small amount of infiltrating epidermoid carcinoma, grade II. Two days later all of this cord was removed by laryngo-fissure. At operation a tiny, 3 mm area of thin leukoplakia was noted on the center of the opposite cord. Anteriorly and posteriorly this cord was normal in appearance and the anterior commissure was clear. Biopsy of this small area of leukoplakia exhibited carcinoma-in-situ as a lesion separate from the invasive carcinoma of the right cord which was well surrounded by normal tissue in all directions.

TREATMENT

When we encountered our first case in 1948 it was necessary to draw upon the experiences of those working in other fields to determine our management of this lesion. Most of these experiences have been with carcinoma-in-situ of the cervix and the majority of the writers were convinced that invading carcinoma would ultimately develop. The length of time necessary for invasion to occur in cases of carcinoma-in-situ was thought to be quite variable; Pund, et al[10] concluded that carcinoma-in-situ averaged five to six years before becoming invasive. Therefore, we concluded that this lesion must be treated practically the same as an invasive one. Because this patient's visible lesion was a coating of leukoplakia involving all of one cord from anterior to posterior commissure, and because multiple areas of carcinoma-in-situ were found in the tissue stripped

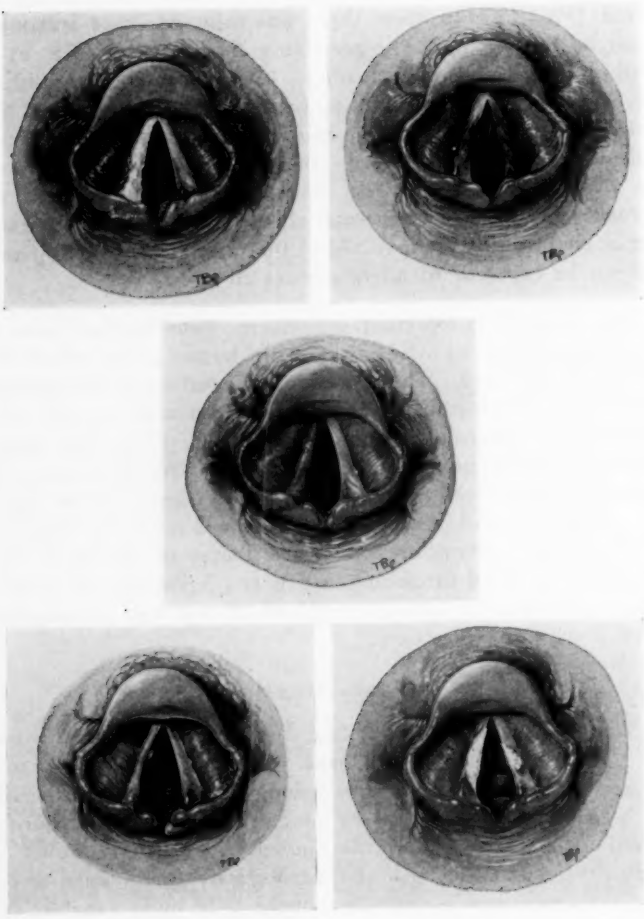

Fig. 5.—Thick leukoplakia with carcinoma-in-situ.

Fig. 6.—Thin and punctate leukoplakia over thickening with carcinoma-in-situ.

Fig. 7.—Diffuse red swelling with carcinoma-in-situ.

Fig. 8.—Localized red swelling with carcinoma-in-situ.

Fig. 9.—Both cords involved by leukoplakia with carcinoma-in-situ.

TABLE III.

TREATMENT AND RESULTS.

	NUMBER	CONTROLLED	RESIDUAL CA-IN-SITU	RESIDUAL KERATOSIS
Stripping	1	1		
Laryngo-fissure only	2	2		
Radiation only	3	3		
Radiation and stripping	7	5	2	2

from the surface, it was decided that just this stripping could not be considered as having removed all possible areas of carcinoma-in-situ. Similarly it did not seem that a laryngofissure could insure removal of all potentially invasive tumor. It was decided to give this larynx a cancericidal course of deep x-ray irradiation. We were supported in this decision by the satisfactory experiences of most radio therapists in treating carcinoma-in-situ of the cervix.

This patient (Case 1) received a tumor dose of 3936 roentgens in 30 treatments. In 33 days his larynx was clear of leukoplakia or other lesion. One year and nine months later, in July of 1950, after having started smoking again for a few months, leukoplakia reappeared in the middle of the opposite vocal cord. This disappeared in one month on again discontinuing smoking. This cycle of events was repeated in March of 1951 and again in May of 1952. On May 16, 1952 the left cord, the site of the original leukoplakia with carcinoma-in-situ, was normal, but thick leukoplakia was stripped from the anterior and posterior thirds of the right cord. This was reported as keratosis with no carcinoma-in-situ. After four and a half years following x-ray treatment there is no evidence of invasive carcinoma or carcinoma-in-situ.

Since the first case, nine additional patients in our series have been treated by fractional deep x-ray irradiation receiving a tumor dose of 3690 to 4305 roentgens (Table III). In seven of the total of ten patients so treated the superficial lesion was first stripped away. Seven of the ten patients treated with x-ray now appear to be clinically controlled; the longest elapsed time following treatment being

53 months and the shortest, five and one-half months. Two patients had a recurrence of carcinoma-in-situ appearing 6 months and 16 months respectively; in two tiny, 2 mm nodules in each case. Following endolaryngeal removal of these nodules the larynx has remained normal in appearance to the present time, for ten months in one case and for six months in the other. The tenth patient completed his course of x-ray treatment only four weeks ago. The first patient treated with irradiation has residual keratosis but no carcinoma-in-situ.

Two patients were treated by laryngo-fissure only. After 31 and 16 months respectively both are apparently controlled. Stripping only, was done in one patient and this larynx has remained normal for the past eighteen months.

COMMENT

We have leaned toward radiation therapy because of the general radiological experience with carcinoma-in-situ, and the ability of this modality to reach all possible sites of involvement by this early and superficial malignancy. Our experience suggests that carcinoma-in-situ of the larynx can usually be controlled by irradiation, but that recurrence after this type of treatment does develop in some cases.

Conservative surgical treatment by stripping, or even laryngo-fissure, presumably has limitations to the completeness with which all possible foci of carcinoma-in-situ can be removed. Nevertheless, our three cases having only this type of therapy remained controlled.

Combination of stripping with radiation has been elected most frequently with the underlying concept that the advantage of both forms of treatment would be secured. The stripping was usually done in the extensive lesions before the exact nature of the lesion was known. This obtained complete removal of the superficial lesion and decreased the chance of overlooking any small foci of carcinoma-in-situ. Obviously it is imperative to individually judge each case as distribution of the lesion will influence the degree and type of surgical treatment possible. In all cases we advise complete discontinuance of smoking and vocal abuse. Recently we have prescribed a high vitamin intake.

So far, no firm conclusions can be drawn concerning the ideal treatment that should be utilized and many questions remain unanswered. The case that returned to normal after only biopsy is

provocative. We believe it will require large groups of cases treated with the several possible combinations of radiation or surgery or both of these and observed over long periods of time, before any final conclusions can be drawn. To this end we suggest that a separate tumor registry for this lesion be established.

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XXXVI

CHORDOMA—FINAL REPORT AND RE-EVALUATION OF TREATMENT

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NEWARK, N. J.

In 1947 Imperatori reported a case of chordoma of the cervical vertebrae before this Society, with a very promising result. The purpose of this paper is to follow-through the subsequent progress of this same patient. To read the paper in its entirety would take up too much time.

There is no need for my going into the histopathology of chordoma, as this has been very clearly defined by Imperatori in the first part of this paper.

The paper is divided into three parts:

Part I is a report in detail by Dr. Imperatori as published in the Transactions of the American Laryngological Society in 1947; with comments in several instances as noted in brackets.

Part II concerns the subsequent progress of the case from Imperatori's record to the time the patient came under my observation, also with comments.

Part III is the subsequent progress of the case while under my observation until his demise. The rapid development of the final symptoms are facts of interest.

PART I

Imperatori reported before this Society in Volume 68, in 1947, a very interesting paper on "Chordomas of the Cervical Region." I shall include his paper with my own so as to bring the history of this case to its conclusion.

To quote Imperatori's paper: "These rare tumors that are vestigial remnants of the notochord[1] (an axial structure characteristic of all vertebrates), are usually located at those areas representing its

extremities, viz., the spheno-occipital synchondrosis and the sacro-coccygeal region. Rarely are they found in the cervical areas.

"Normally,[2] portions of the notochord persist in the intervertebral discs as the central mucoid cores or pulpy nuclei (nuclei pulposi).

"Virchow,[3] in 1857 described small jelly-like tumors occurring in the region of the dorsum sellae and named them "ecchondrosis physaliphora." These were attributed to islets of degenerated cartilage from the spheno-occipital synchondrosis.

"Fabricius reported,[4] a case history of a patient with a chordomatous growth of the nasopharynx and pharynx, who was operated upon twice, the second operation being four and one-half years later, and who remained well without further recurrence. Complete details are not given.

"Syme and Cappell,[5] described in some detail such a tumorous growth. It was located on the posterior pharyngeal wall and grew upward toward the nasopharynx and downward toward the larynx, being attached to the 2nd, 3rd and 4th cervical vertebrae. The chief symptoms were pain in the head and neck, stiffness in the neck and difficulty in swallowing. The patient was operated upon in July, 1926, and by January, 1927, a second operation became necessary, but was unsuccessful. The approach was externally. There was no lymph node involvement and no invasion of the adjacent structures observed at the operation in January. However, one lymph node was found to be involved at the second operation.

"Andre-Thomas and Villandre,[6] reported a case, and Cappell,[7] reported two such tumors attached to the cervical vertebra.

"Chiari[8] reported a cervical chordoma which was attached to the surfaces of the borders of the 4th and 6th cervical vertebrae.

"Definitely, chordomas of the pharyngeal and mid-cervical areas are of such uncommon occurrence that they warrant recording, and such a report will be included in this communication. [The ultimate result of this case will be reported by the present author.]

"Ridpath[9] states these tumors 'have been reported as occurring in various locations in the following order of frequency: In the sacro-coccygeal in ninety-one cases; in the spheno-occipital region in fifty-two cases; in the cervical portion of the spine in twelve cases, and in other locations, rarely.'

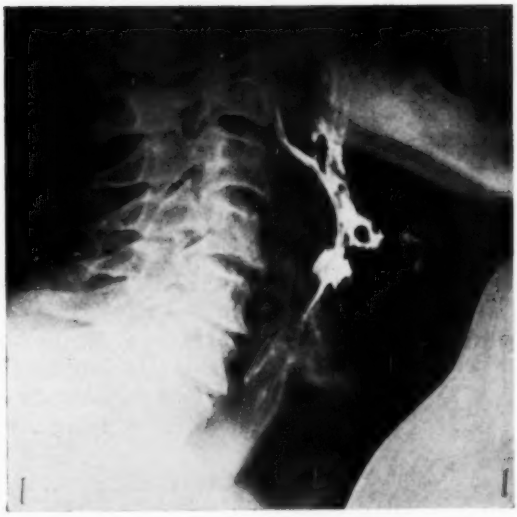

Fig. 1.—Lateral roentgenogram, November 7, 1945. Shows a shadow extending forward from the cervical vertebral region. (From Imperatori's paper)

"Gavin Livingstone[10] groups these tumors into three classifications: 'Type 1. The small tumors called by Virchow " ecchondrosis physaliphora." They occur in two per cent of postmortem examinations and are composed of the same type of cells as the nucleus-pulposus of the intervertebral discs.

" 'Type 2. The malignant tumor in which active mucin-producing cells predominate and in which recurrences and metastases occur.

" 'Type 3. The main feature of this type is the production of cells which resemble cartilage and the tumors are usually encapsulated, and do not recur.'

"A great many of the cases reported as occurring in the cervical area have been high, usually arising from the 1st and 2nd cervicals, and they were usually associated with growths in the spheno-occipital synchondrosis. Very complete and detailed articles by Owen, Hershey

and Gurdjian;[11] by C. M. Haas[12] and by Adson, Kernchan and Waltman[13] have dealt mostly with cranial and high cervical located tumors.

"Many authors have expressed varying opinions as to the cause and origin of these tumors.

"It would seem that there is a unanimity of opinion that they originate from displaced notochordal remnants. The cause of their growths has not been satisfactorily explained for Stout[14] reports a maxillary chordoma in a day-old infant, and the patient on whom this report is being made is 63 years old.

Diagnosis. The exact diagnosis cannot be made without biopsy. However, a slowly growing tumorous mass appearing in the oro or laryngopharynx, producing pressure symptoms on the adjacent nerves of the cervical plexus, disturbance in breathing and deglutition, running an afebrile course, and on palpation having a tense jelly-like feel, should be suspected as possibly being a chordoma of this region.

"X-ray will show a bulging shadow into the pharynx of varying density. With barium the shadow is very clearly outlined. Also, any involvement of the bodies of the vertebra and the intervertebral discs may be determined. The amount of obstruction to the food and air passages may also be evaluated.

"Aspiration biopsy will, very likely, give negative results, excepting that if the tumor mass contained contents that were more fluid than semisolid, a diagnosis might be arrived at. Cysts, retropharyngeal abscess, etc., etc., must be differentiated.

"Stout[15] epitomized the microscopic findings by stating that chordomas are characterized by the so-called physaliferous cells which are polyhedral with a small nucleus and cytoplasm much distended with glycogen and vacuoles containing a mucin-like material. This gives to the tumor its characteristic jelly-like gross appearance.

"The desiderata necessary to diagnose a chordoma has been postulated by Linck:[16] 1. Syncytial giant cells. 2. Curious concentric nest of cells. 3. Nuclear vaculation possibly filled with glycogen. 4. Physaliferous cells (nucleus is located peripherally and the cytoplasm is filled with mucinous globules.) 5. Irregular traveculae of tumor cells separated by abundant intercellular mucinous matrix.

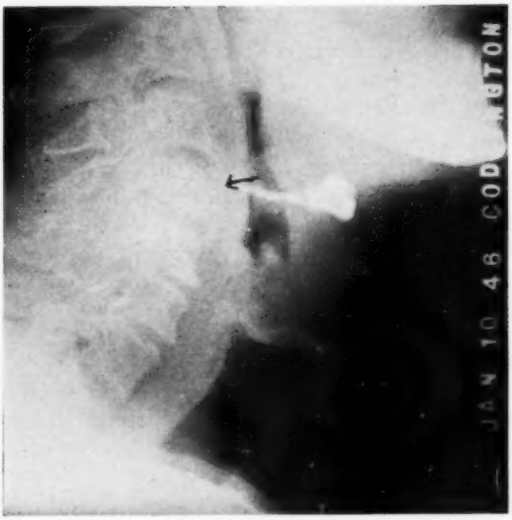

Fig. 2.—January 10, 1946. There is no bulge in this roentgenogram of the posterior pharyngeal wall, but you do see a beginning bridging of the 3rd and 4th cervical bodies and a rarification of the 4th cervical. (From Imperatori)

"It has been refuted by many who have studied these tumors that the substance within the much distended cell is glycogen, the chemical test having been confused by the fixative.

"*Treatment.* This tumor is invasive and very resistant to roentgenotherapy. Its anatomical situation is such that the external surgical approach is difficult. [This is not so, as I will show later in Part III of this paper. H.B.O.]

"Suspension laryngoscopy gives an excellent exposure of the pharynx and laryngopharynx, and should be the method of choice in the excision of such a tumor, located between the 3rd and 6th cervicals.

"The mucous membrane, submocosa and pharyngeal aponeurosis may be dissected from the tumor, and excepting where it is

attached to the bodies of the vertebra, may be removed intact. These tumors are usually well encapsulated.

"The following case report is briefly detailed. W. C., male, aged 62, American, referred by Dr. Brandon of New York City. The patient states his father died when 76 years old from a tumorous growth of the rectum, that necessitated a colostomy. Up to June 1945, he had no serious illness. About that time, he noticed that he was having slight difficulty in swallowing, but attributed this to the trouble he was having with his teeth. Recently, the difficulty in swallowing increased so that he experienced a sensation of complete obstruction, and particularly so when the larynx is drawn up by the hyoid muscles during the act of swallowing. He states that it feels as though a "ball valve" closed.

"During the past few months, he has lost considerable weight (40 lbs.) and his strength and vigor have diminished accordingly. There have been no pain or pressure symptoms on any of the adjacent nerves.

"*Physical Findings*: The general appearance of the patient is that of an emaciated elderly male, very apprehensive, and somewhat non co-operative. All of his teeth are in a state of partial or total decay, gums swollen and inflamed, and many areas of superficial ulceration of the mucous membrane of the mouth. The epipharynx and nostrils were negative.

"By indirect laryngoscopy, there is seen an obstructing mass slightly to the right of the mid-line. It is smooth in outline, covered by the normal mucous membrane of the pharynx and without irregularities or ulcerations. The growth is approximately at the lower level of the epiglottis and extends downward on the posterior wall. The larynx, including the epiglottis, is seen by indirect laryngoscopy, when the chin is elevated and the neck extended. The vocal cords were visualized in their entirety to the anterior commissure. The ventricular openings appeared to be normal. External palpation showed the larynx movable but not freely, and no creptitations were felt on the spinal column. The mass within the hypopharynx cannot be palpated externally with any degree of certainty as to its outline. There were no enlarged lymph nodes. Digital palpation reveals that the hypopharynx is almost completely obstructed. Temperature range was 98 to 99 degrees.

Fig. 3.—Roentgenogram taken November 18, 1946. There is nothing to show any recurrence of the tumor which was removed, but there is increase of the calcification in the 3rd and 4th cervical bodies bridging these two bodies. (From Imperatori)

"*Tentative Diagnosis*: Soft tumor of the hypopharynx, possibly a cyst. Advised Roentgenogram, aspiration biopsy and biopsy.

"The x-rays made by Dr. Ramsay Spillman of New York showed the extent of the tumor—from the 3rd to the 6th cervical and protruding forward. The meager report was helpful in that it showed the extent of the obstructing mass but nothing else, excepting in a negative way—to state that there was no osteomyelitis of the spine, or tuberculous.

"Aspiration of the tumor was productive of no data.

"Under local anesthesia, a biopsy was done into the center of the tumor. The opening into the pharyngeal tissue was less than 5 mm. There was no reaction following this procedure. The patient was given sulfadiazine, and after two days was permitted to leave the

hospital in order to adjust his affairs, have his decayed teeth removed and return to the hospital within two weeks.

"The pathological report of the biopsy was somewhat confused but after consultation with several pathologists, it was decided that it was a chordoma and that this would be confirmed by the gross and microscopical examination of tumor, when it was removed.

"*Operation*: On November 19, 1945, at the Manhattan Eye, Ear and Throat Hospital, the tumor was removed with the patient in the suspension laryngoscope. Ether anesthesia. The procedure consisted of making a transverse incision of the mucous membrane and subadjacent tissues over the greatest prominence of the tumor, and separating a rather thin-walled capsule of the tumor from the adjacent structures. The tumor was attached to the 4th cervical region and it was necessary to use sharp dissection in order to free it. The tumor mass was delivered from its bed almost intact. The posterior attachment was somewhat ragged. The was comparatively little bleeding and this was controlled by pressure. There were no distinct bleeding points, the bleeding being of the capillary type.

"A nasal feeding tube was inserted and the patient returned in very good condition. Within 18 hours, the patient was out of bed for a short period (five minutes) and within five days, the feeding tube was removed. He was able to leave the hospital November 25, 1945.

"For the four days after operation, he received 25,000 units of penicillin every three hours.

"*Pathological Report*: Gross: Gelatinous mass enclosed in a fibrous capsule approximately 5 cm x 4 cm x 2 cm with fibrous bands running through it and attaching themselves to the capsule. It has a slightly reddish color and a peculiar odor.

"Microscopic: Sections show that there is a tumor made up of a peculiarly large, polygonal cells which are arranged, sometimes in cords, and are surrounded by mucoid material. The individual cells sometimes attain a gigantic size and some are vacuolated, producing a physaliferous effort. The tumor grows diffusely and tends to infiltrate. This seems to be a characteristic chordoma in one of the less common situations.

"Diagnosis: Chordoma of the Hypopharynx. A. A. Eggston, M.D. Pathologist, Manhattan Eye, Ear and Throat Hospital; Arthur P. Stout, M.D. Consultant.

"*Conclusions*: Herewith is a report of a chordoma situated in the cervical region (3rd to 6th vertebrae) with diagnostic procedure and method of treatment. There has been no recurrence to date, and the patient is in good physical condition."

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PART II

The second part of this paper pertains to the follow-up, studies and operations from Dr. Imperatori's own record which the patient gave me, having received the same from Dr. Imperatori at the time of his retirement from practice. The following, therefore, are his progress notes from 1945 until the patient's first visit to me on January 21, 1950.

"February 23, 1946—there is no evidence of any recurrence; the larynx and piriform sinuses are clear. X-rays taken January

Fig. 4.—Roentgenogram taken December 15, 1947, shows rarification of the 3rd and 4th cervical vertebrae, with feeding tube in position following laryngectomy.

10, 1946 revealed absence of any growth. The patient continued in good condition until January 21, 1947 when he complained that he was having some difficulty in swallowing, especially on eating meat. The examination at this time revealed there was a mounting up of tissue in the pharynx, just above the right arytenoid, about the size of a small olive. The following September 1947, patient had the sensation of obstruction; the growth beginning to fill up the pharynx, and also in front of the larynx.

" 'X-rays taken show evidently a local recurrence of the tumor in the same location as before. There is a rounded mass encroaching on the entrance to the larynx, not only behind, but in front, displacing the epiglottis backward, and there is only a filiform channel as visualized in profile that gives any air-way. In the A.P. projection, the tumor lies on the right side. There is no evidence of involvement in the chest." (R. Spillman, M.D.)'

Fig. 5.—Shows the pharynx outlined with barium following laryngectomy.

"December 11, 1947, the patient's weight was 145 pounds and he was admitted to the Manhattan Eye, Ear and Throat Hospital, and under general ether anesthesia attempted a suspension laryngoscopy, which gave a fair exposure but not sufficient to operate properly; the patient had considerable difficulty in breathing during the anesthesia. It was decided that any further attempt would be futile, that the growth should be removed by subhyoid or lateral pharyngotomy. A preliminary tracheotomy in the lower trachea was done. There were no post-operative complications.

"December 15, 1947, an attempt was made to remove the growth through a subhyoid pharyngotomy, but found this could not be done without removing the larynx, which was done. The growth was large and attached to the bodies of the 4th, 5th and 6th cervical vertebrae. These were eroded. The lesion was soft and mushy, most of the mucous membrane on and over the tumor was saved. The trachea was sutured to the skin. There were no post-operative complications.

" 'The pathological report on section shows striated muscle, dense fibrous tissue, granulation tissue, area of myxomatous degeneration, areas of necrosis. Some large infiltrating syncitial like masses of cells, some stratified squamous epithelium apparently esophageal; some sero-mucinous glands. Very little tumor tissue present, some cells show mitosis, and some contain globules, and are physaliferous; an area shows acute inflammatory reaction. Diagnosis: Recurrent chordoma.' (Dr. Eggston.)

"The nasal feeding tube was removed December 24, 1947, and on the 29th of December, the patient was discharged from the hospital. He was seen a number of times in the office during the month of January 1948; by February 14th he had gained in weight, now 155 pounds, and was swallowing normally.

"April 1, 1948, x-rays were taken by Dr. Spillman who reports as follows: 'There is no thickening of the retropharyngeal and retroesophageal tissues in the neck as shown by barium examination, nor do I find anything abnormal in the chest. The changes involving the 4th and 5th cervical vertebrae are very interesting. At the original examination, November 7, 1945, I reported a little decalcification of the ventral surface of the 4th cervical body, and had no conception of its presumable significance. On November 18, 1946, I noted a bony density apparently originating in the 3rd and 4th cervical bodies, and bridging the ventral surfaces. Since then the process has extended both up and down, and a tongue of bone casting a shadow about 5 x 7 mm extends down into a notch at the upper ventral angle of the 5th body, and a similar small bony mass is seen just ventral to the lower half of this body. If this process is related to the chordoma, it shows a spontaneous calcification.'

"January 10, 1949, the patient is swallowing very well, the laryngectomy stoma looking very good and apparently no recurrence." [This Dr. Imperatori's last note concerning the patient.]

Part III

I shall now take up from here when the patient was referred to me by Dr. Sandella of New Brunswick, New Jersey, on January 21, 1950.

My examination showed a well-developed white male, aged 67 years, with a laryngectomy stoma, having difficulty in swallowing, and choking at times, which had been gradually becoming worse.

Fig. 6.—Roentgenogram taken January, 1950, shows definitely an increase of this rarification of the 3rd cervical vertebrae.

On further examination I found on the right side of his pharynx, a swelling, extending downwards for a distance of 3 cm. Palpating this swelling revealed that it was hard and extending from the third cervical vertebrae downward to the fourth and fifth. No palpable lymph node enlargment was noted.

X-rays taken at this time accorded with the above; a diagnosis was made of a recurring chordoma of the pharynx, and a lateral pharyngotomy was indicated.

February 24, 1950, the patient was admitted to the Presbyterian Hospital in Newark, and on March 2nd a lateral pharyngotomy was performed under avertin-ether anesthesia. Incision was made along the anterior border of the right sterno-mastoid muscle, at the lower part of which it was extended mesially to make a skin flap to be used in the closure. All vessels coming off anteriorly from the large vessels in the neck were ligated and severed. The thyroid gland was isolated. Fortunately we found a piece of the right horn

of the thyroid cartilage which had not been removed at the time of the laryngectomy; this served as a guide. The attachment of the inferior constrictor to this was severed, thereby exposing the pharyngeal aponeurosis. This was incised and the growth visualized. The growth extended over the bodies of the third and fourth cervical vertebrae, and was removed without difficulty. At the same time some material contained in the growth was noticed in the fourth cervical foramen. This predisposes to further recurrence. The pharynx and overlying structures were closed, and the patient returned to his room. The only difficulty encountered in the operation was that all the normal landmarks for this type of operation had been eradicated.

The pathological report of the growth is as follows "The growth is 3 x 2 x 1 cm. It appears to project from a mucous membrane, forming crypts. Microscopically it shows atypical large cells with large irregular hyperchromatic nuclei separated by collagen fibers and vacuolated in areas. It is generally quite a vascular growth. In places it contains hematogenous pigment. There are areas of bone formation. Diagnosis: Chordoma of the pharynx." (S. A. Goldberg, M.D.)

The post-operative period was uneventful and the patient was discharged from the hospital on March 15, 1950. He progressed nicely until December 21, 1951, when he came to the office complaining of a stiff neck. In looking into the possible cause of this condition, and upon questioning him, he disclosed the fact that during the past month he had been doing some painting in his home, and thought he had strained some muscles in his neck, and this might be the result. He also stated that he had some pain in his shoulders before he finished painting but did not think much of it until after painting the ceiling which caused him to have severe pain in his neck. The pain had increased with muscle spasm for the past two weeks. This was followed with difficulty in elevating his arms, especially when he attempted to lift some coal or ashes. At the present time he has noticed some unsteadiness while walking and is afraid to go out, also that his shoulders are getting numb.

X-rays taken on the same day, December 21st, are as follows: "Examination was made of the cervical spine in the antero-posterior, lateral and oblique projections. Examination of the cervical spine shows the normal lordotic configuration of this portion of the spine.

Fig. 7.—December 21, 1951. Roentgenogram shows marked rarification of the 3rd cervical vertebrae with some involvement of the 4th; also the lower arrow shows a slight amount of pantopague in the canal.

The vertebrae are all in good alignment. There are changes seen in the mid-cervical region. The body of the fourth cervical segment shows irregular areas of decalcification and sclerosis obliterating and distorting the normal cancellous bony structure throughout the entire body of this vertebra. Similar but less marked changes are seen in the body of the sixth cervical segment. There is a rather extensive intervertebral bridging extending along the anterior border of the vertebrae from the third to the sixth segment inclusive. This is rather thick in the mid-cervical region and has immobilized this portion of the spine. There are also arthritic changes noted in the lower cervical region and partial collapse of the intervertebral spaces between the fifth and sixth as well as the sixth and seventh. There is no collapse of any of the vertebral bodies. The antericular facets posteriorly show very little evidence of pathology.

"The changes in the fourth cervical body could be the result of direct extension of neoplasm into the bone substance. However,

I am inclined to believe that there would be greater changes noted probably with collapse of the body if the bony structure were involved. The bridging anteriorly obscures much of the bony detail and could give the appearance seen here because of the superimposed density.

"While there is considerable osteo-arthritis with lipping of the faces of the bodies of the vertebrae throughout the entire circumference of the superior and inferior faces, the arthritis does not appear to be advanced enough to cause the symptoms in this case. Some of the lipping is seen posteriorly extending beyond the limits of the bodies of the vertebrae but this would not cause any marked compression of the cord. There are some changes in the vertical diameter of the intervertebral foramen as the result of the collapse of the two interspaces which may be contributing to the symptoms. Radiating pain over the shoulders and down the arms is frequently seen with this amount of spondylitis, and I believe it could very well explain the symptoms.

"There is some soft tissue thickening on the posterior wall of the lower pharynx just anterior to the fourth cervical vertebra but no soft tissue mass can be seen. The question, therefore, of extension of a neoplasm into the fourth cervical segment in this case cannot definitely be determined on the basis of this one examination. There are changes seen in the bony structure which could be the result of neoplasm, but I believe that after the length of time since the original lesion this may be the result of the changes around the cervical spine. One would expect a certain degree of collapse of the body if this were all neoplastic tissue." (F. P. Carrigan, M.D.)

On December 22, 1951, the patient was admitted to the Presbyterian Hospital. The report of the examination by the neurosurgeon, Dr. Richard Swain, is as follows: "Diminution of sensation in both arms up to the top of the shoulder. Marked motor weakness in some areas. Biceps reflexes weak. K.I. hyperactive. Ankle clonus present bilaterally. Babinski plus bilaterally R to L. C-1, C-2, C-3, C-4, seem to be clear. There is probably some cord compression as well as root involvement of C-3 to C-7 or T-1. The question arises of a laminectomy for exploration and possible decompression of roots and cord in the involved area. This would depend somewhat on his life expectancy as to prognosis."

Fig. 8.—December 21, 1951. Shows x-ray of the myelogram with pantopaque material.

On December 24 a cervical myelogram was done and the examination of the roentgenogram made with the patient in the Trendelenburg position shows the opaque medium to be at about the level of the upper border of the C-6. The upper border is somewhat irregular in outline. This confirms by fluoroscopic observation that there is a block in the spinal canal at about the level of upper border of the C-6.

On December 25 the patient became much worse with difficulty in breathing, both legs were paralyzed, and laminectomy was immediately performed.

The preoperative diagnosis was cervical chordoma compressing the spinal cord at the level of the third and fourth cervical vertebrae.

A midline incision was made in the cervical region; the muscles were separated from the spine and laminae of the second, third and fourth cervical vertebrae. These laminae were removed with rongeurs exposing the dura. The tumor mass removed as far as possible, but the bones of the bodies of the vertebrae were evidently infiltrated by the tumor, preventing removal. The dura was opened in the midline. The wound was closed with interrupted sutures, muscles

Fig. 9.—Photograph of post-mortem specimen showing fracture of the 3rd cervical and involvement of the 4th cervical vertebrae.

and skin with black silk. The patient was returned to bed. (Dr. Swain.)

Findings: A tumor mass was present in the vertebral canal anterior to the cord. The spinal cord had been greatly compressed by the extradural tumor.

December 26, at 2:45 a.m. the patient ceased breathing.

Post-mortem findings: Chest—Lateral adhesions on both sides, and on the left side there is an adhesion to the diaphragm; Lungs—Pericardial sac clear; Heart—mitral valve contains small atheromata; Abdomen—free and clear. Liver not enlarged. Spleen negative. Kidneys, adrenals, bladder and prostate negative.

On opening the cervical canal the cord at the level of the third cervical vertebra was markedly thinned, soft and mushy. The lining of the canal in the area was roughened. On section the anterior horns were discolored.

The anatomical diagnosis was: Respiratory collapse, contributary cause compression of the cervical cord at the level of the third cervical vertebra.

Fig. 10.—Photomicrograph of the tumor from post-mortem specimen shows atypical large cells with large irregular hyperchromatic nuclei separated by collagen fibres and vacuolated in spaces.

The lining of the canal in the region of the third cervical vertebra ventral to cord, was composed of atypical large cells with large hyperchromatic nuclei separated by hyaline substance. The cells were pleomorphic, and a very occasional one contains a vacuole. The bony substance in the body of the third cervical is infiltrated and eroded out by these cells. These fill the marrow spaces and replace the marrow elements. There are areas of necrosis with neutrophilic infiltration. The intervertebral cartilage in the area is markedly degenerated. In the cord below the compressed area practically all of the motor cells are degenerated, very few remaining which contain nuclei.

The ependymal canal is compressed and practically obliterated. There are scattered areas of hemorrhage and hematogenous pigment. The Nissl's granules are gone from practically all of the motor cells. Rarely there is one which still contains a few Nissl's granules. The

vessels of the cord are congested so that the Virchow Robin spaces are nearly obliterated. Higher up, where the cord is partially compressed, the general outline of the cord is altered. There are large areas of hemorrhage. At the compressed area the cord is completely disorganized and contains irregular vacuolated areas and small areas of hemorrhage. The vessels are congested. Motor cells are not seen.

The histological diagnosis is chordoma of the body of the third cervical vertebra producing a pathological fracture and pressure upon the cord, causing atrophy of the cord in that area and paralysis of the upper and lower extremities; acute suppurative osteomyelitis of the third cervical vertebra. Atrophy and hemorrhage of the cord from the third cervical down. Early adenocarcinoma of the prostate.

TREATMENT

Chordomata are resistant to irradiation. Under treatment (Part I) it is mentioned that, "its anatomical situation is such that the external surgical approach is difficult; and it is further stated that suspension laryngoscopy gives excellent exposure and should be the operation of choice in the excision of such a tumor located between the 3rd and 6th cervicals."

With a recurrence December 11, 1947, the author states (in Part II) "that a suspension laryngoscopy did not give sufficient room to operate properly." [At this time I had suggested that a lateral pharyngotomy be performed. H.B.O.] On December 15, 1947, a subhyoid pharyngotomy was performed, but it having been found that this could not be done without removing the larynx, laryngectomy was done.

The patient came under my observation in January 1950, (Part III) with a recurrence in the same region, and the only operation that could be done was a lateral pharyngotomy which gave ample space to operate, notwithstanding that many of the landmarks for this type of operation were gone due to previous operations.

My re-evaluation indicates that the best procedure for this type of chordoma is a lateral pharyngotomy, thereby saving the patient's larynx.

Irrespective of what type of operation was done, the body of the vertebra was involved even from the time of the first operation; painting and stretching his neck caused the patient to sustain a path-

ologic fracture of the third cervical vertebra causing compression of the cord, and death.

SUMMARY

A complete report is given, of a patient with a cervical chordoma, including the various operations and findings, from the inception of the tumor to the patient's demise.

Part I concerns the first report by Dr. Imperatori with his comments on treatment and references; Part II, his follow-up of the patient with subsequent operations for recurrence; Part III, the record of the patient since he came under my observation, with operations, follow-up and post-mortem findings.

CONCLUSION

Chordoma of the pharynx is rare and usually fatal. It is usually of slow growth and rarely metastasizes: but apparently it invades the surrounding bones with destruction of the adjacent bony structures.

Involvement of the vertebrae was demonstrable in the roentgenograms, but apparently the roentgenologists did not associate the lesion in the pharynx with the changes in the bone, although they did mention the fact that arthritic changes had taken place.

The diagnosis is not difficult when the lesion projects into the pharynx. In the early stages the mass must be differentiated from sarcoma, and fibroma. They are all smooth, round, firm growths which erode bone. Absolute diagnosis is by biopsy.

224 DELAVAN AVE.

XXXVII

NEURILEMMOMA (SCHWANNOMA) OF THE LARYNX

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There has been a great deal of confusion regarding neurilemmomas, neurofibromas and other tumors arising from nerves and nerve sheaths. Stout[1] and Ewing[2] seem to be in very close agreement, in classifying and in differentiating neurilemmomas and neurofibromas. We take our description from Stout. He classifies neurilemmoma under "Neuroectodermal Neoplasms—Supportive Tissue Type—Benign," and gives twenty names which the tumor has been called. Among these are: neurilemmoma (Stout), neurinoma (Ewing), acoustic neuroma, schwannoma and perineural fibroblastoma.

We quote from Stout: "This is the characteristic solitary, benign, encapsulated tumor occurring in the peripheral, cranial and sympathetic nerves; and arising from the sheath of Schwann. . . . While usually solitary and spontaneous, neurilemmomas may be multiple, and can be found in Von Recklinghausen's Disease. They may arise at any age, in either sex, and probably in all races from any nerve or nerve root that has a schwannian sheath. . . . These tumors are benign and always encapsulated. They rarely recur after removal even if the capsule is left behind. It is questionable whether or not a malignant tumor ever develops from them.

"Histologically the picture is characteristic. Inside the sheath the tumor tissue is composed of two parts intermingled but quite sharply defined one from the other. The nuclei often show a tendency to be aligned in rows with intervening spaces without nuclei. This is often called palisading of nuclei. In the neurilemmoma this arrangement of cells, nuclei and fibers often assumes an organoid appearance suggesting an exaggerated tactile corpuscle sometimes called a Verocay body. . . . "

In the literature regarding laryngeal conditions these tumors are called neurofibromas in most instances. From the gross and

microscopic descriptions given we feel that some are neurilemmomas and some are neurofibromas. We quote again from Stout in order to justify this opinion.

"The neurofibroma is characterized by a diffuse proliferation of the peripheral nerve elements in contradistinction to the neurilemmoma which is the encapsulated tumor. It may occur as a solitary localized phenomenon but is much more commonly observed as part of the condition known as multiple neurofibromatosis or Von Recklinghausen's disease. . . . The neurofibroma grows in a number of different ways. A common one is at the end of a nerve. This can produce isolated small nonencapsulated nodules. . . .

In a search of the American literature we have been able to find reports of 21 tumors of the larynx diagnosed as neurofibromas, Schwannomas or neurilemmomas. We have not included those reported as being associated with Von Recklinghausen's disease, nor eight cases reported from foreign literature by Fisher and Odess,[3] nor 22 laryngeal neurinomas reported in abstract in the 1950 Yearbook by Helg[14] of Lausanne, Switzerland. These latter groups have been omitted because sufficient descriptive information was not available to attempt differential classification.

Study of the gross and microscopic descriptions of the tumors reported in the American literature reveals that ten of the 21 are described as polyps or tumors of the vocal cord margin. These do not mention encapsulation and in some instances mention suspicion of malignancy because of recurrence after removal. We believe that these are neurofibromas rather than neurilemmomas according to the classifications of Stout and Ewing.

These ten cases are reported as follows:

1. New and Erich[4] in a study of 722 benign tumors of the larynx list one neurofibroma from the middle third of a vocal cord.

2. Jackson and Jackson[5] list four neurofibromas of the vocal cords without detailed description.

3. New and Devine[7] found one marginal cord tumor and one from the posterior surface of the arytenoid among 19 neurofibromas of the nose and throat. Four of the 19 tumors were associated with Von Recklinghausen's disease, not including either of the laryngeal tumors. We are listing one of these as a neurofibroma and one as a neurilemmoma, perhaps incorrectly.

Fig. 1.—Photograph of Tumor.

4. Perlman, Friedman and Appel[8] restudied the microscopic sections of a series of fifty tumors originally diagnosed as fibromas of the larynx and found four vocal cord polypi which in their opinion were neurofibromas. They reported that these tumors were not encapsulated, had invasive characteristics and a tendency to recur after removal.

The tumors which we believe to be neurilemmomas are reported as follows:

1. Jackson and Coates[6] report as a neurofibroma a tumor of the larynx in a patient who had carcinoma of the thyroid but make the statement that the microscopic picture looks like a neurilemmoma.

2. One of the two tumors reported by New and Devine as listed above.

3. Vail[9] in 1933 described a large Schwannoma removed from the aryepiglottic fold. Incidentally, this is the location of five of the eleven neurilemmomas found.

4. Thomas Smith[10] reported three large tumors of the aryepiglottic fold, two of which it was necessary to remove by laryngofissure and the third by external hypopharyngotomy. He described them as solitary, benign, encapsulated, slow growing and having no tendency to recurrence. He also mentioned that it was difficult or

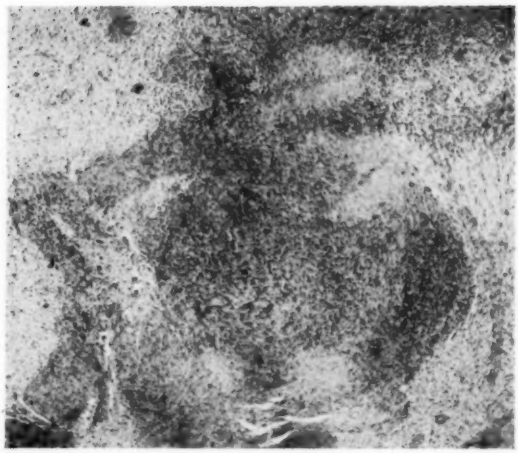

Fig. 2.—Low power photomicrograph of neurilemmoma of the larynx. Note Verocay bodies and division into Antoni type A and Antoni type B tissue.

impossible to obtain biopsies by direct laryngoscopy because of the consistency of the tumors.

5. VanLoon and Diamond[11] reported a large encapsulated tumor causing gross swelling of one side of the larynx with obliteration of the ventricle. Repeated biopsies yielded only mucosa overlying the tumor. It was removed by laryngofissure and diagnosed as neurofibroma. We believe that this was a neurilemmoma according to the classification of Stout and Ewing.

6. Hoover[12] in 1940 reported a series of benign tumors of the larynx and described one large solitary neurofibroma of the aryepiglottic fold removed through an external approach. Detailed microscopic study was not reported but it is classed as a neurilemmoma because of its clinical similarity to others.

7. Oliver, Diab and Abu-Jadeb[13] reported an encapsulated large neurofibroma of the aryepiglottic fold removed perorally and mentioned that it was too hard to punch with laryngeal biopsy forceps.

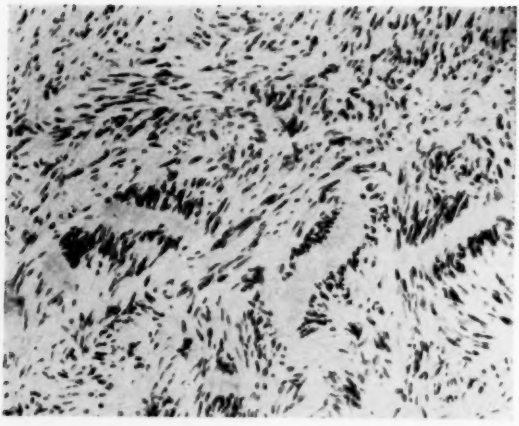

Fig. 3.—High power photomicrograph of neurilemmoma of the larynx. Verocay bodies.

8. Fisher and Odess[3] in 1949 reviewed the literature and listed nineteen tumors as solitary neurofibromas of the larynx. Eight of these were from foreign literature and not available to us. The other eleven are included in the above review. They reported one small yellowish nodular tumor from the base of a vocal cord and another nodule from the aryepiglottic fold. Their discussion would indicate these tumors to be neurilemmomas.

Neurilemmomas are, at least, very unusual tumors to find in the larynx. Neurofibromas also are unusual. If we include those mentioned above from foreign literature approximately only forty of both types of these tumors have been reported. From the articles read, tumors of the larynx associated with Von Recklinghausen's disease seem to be even less frequent. Holinger and Cohen[15] reported such a case and several others have mentioned them. Neurilemmomas sometimes grow to very large size in the pharynx and in the neck. Those interested in the clinical problems of their treatment are referred to the articles by Viole,[16] Turchik,[17] Koop, et al,[18] and Slaughter and DePeyster.[19]

REPORT OF A CASE

Mrs. E. B., aged 48, was first seen in April, 1948, with symptoms suggesting vocal fatigue and strain. She was not objectively hoarse but her voice was weak. Upon examiation there was no visible tumor in the larynx but the left ventricular band looked full and partially hid the vocal cord. There was an apparent bulging medially of the subglottic area on this same side. The arytenoid appeared to move normally. On repeated examinations this medial bulging of the ventricular band and the subglottic area became more definte. On direct laryngoscopy the mucous membrane of the floor of the ventricle seemed thickened and bulging. Two small pieces were removed with biopsy forceps and a smooth, fairly firm, grayish, domelike lesion appeared in the mucosal defect. This was obviously a tumor too large to be removed through the laryngoscope and an attempt to take a specimen with double-cupped laryngeal forceps was unsuccessful. Following this procedure the tumor could be seen bulging from the ventricle on mirror examination. At laryngofissure the tumor was found between the inner thyroid perichondrium and the lateral mucosal wall of the larynx. It was smooth, encapsulated, roughly ovoid and was readily dissected intact from its bed. It measured approximately 2.75 x 1.5 x 1.5 cm (Fig. 1). The larynx healed readily but with scarring of the anterior commissure and left the patient with a husky voice. There has been no evidence of recurrence during more than three years of operative observation.

The gross specimen consisted of an encapsulated, ovoid, relatively mass 2.5 x 1.7 x 1.2 cm. The external surface had a pinkish pale yellow appearance. Cross section showed semi-translucent tissue varying from pinkish gray in its outer portion to orange at its center.

Microscopic examination of a full thickness section of the tumor shows an extremely thin fibrous outer capsule. The tumor proper has a somewhat lobulated appearance due to wide variation in the density of the tumor. In the denser areas there was seen a tangled and frequently whorled arrangement of moderately cellular fibrous tissue. The cells had spindle shaped nuclei in which there was slight variation in the sign of the nuclei, but not to the extent to be indicative of sarcomatous change. Neither were mitotic figures observed. In several of these lobules there were seen very characteristic "Verocay bodies" as evidenced by parallel lines of palisaded nuclei and bands of

collagenous fibrils that stretched between them, forming meandering ribbon-like zones. It is the presence of these Verocay bodies especially that indicate this tumor to be of neurogenic origin. Between these lobules was extremely loose, almost myxomatous type stroma and here small capillaries were observed (Figs. 2 and 3).

This was a benign type tumor which apparently originated from the sheath of Schwann. This type of growth has a predilection for the acoustic nerve. There was no evidence of malignancy. Diagnosis: Benign Neurilemmoma, "Schwannoma." (W. F. Keller, M.D.)

On a second opportunity to remove a tumor in such a location we would approach the tumor laterally through the thyroid wing rather than through the anterior commissure and hope to leave the patient a better voice.

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XXXVIII

HEMILARYNGECTOMY WITH IMMEDIATE SKIN GRAFT FOR THE REMOVAL OF CARCINOMA OF THE LARYNX

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In recent years greater consideration has been given to preservation of function in the surgical treatment of carcinoma of the larynx. Unnecessarily radical operative procedures are now performed less frequently since experience has shown that adequate removal of moderately advanced lesions often can be effected by technics which do not disturb normal physiologic function. The field of usefulness of laryngofissure has expanded considerably during the past few decades. The operation has been made increasingly radical by supplementing sharp excision with electrocoagulation and by sacrificing more of the cartilaginous structures. However, there are definite limits to the extent of tissue that can be removed by means of a thyrotomy or a classic hemilaryngectomy without directly destroying function or causing danger of collapse of the airway. Relining the larynx with a skin graft has at times been carried out secondarily in order to correct the stenosis induced by exceeding these limits. Hemilaryngectomy with immediate skin graft renders it possible to effect more extensive local removal without destroying the voice or disrupting the airway. A preliminary report on this procedure was made before this Association at its meeting in San Francisco three years ago. The procedure has now been performed in a sufficient number of cases to permit adequate evaluation and the results warrant its inclusion in the armamentarium of the laryngeal surgeon.

Hemilaryngectomy with immediate skin graft for the removal of carcinoma of the larynx evolved from the application of certain basic principles of plastic surgery. Among these is the precept that

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appreciable loss of surface covering or of the lining of a viscus must be restored in order to secure prompt healing with minimal scarring and to obviate contracture. Further, a skin graft should not be used to cover a defect unless there is reasonable assurance that the pathologic process responsible for the loss has been completely eradicated. Experience with the surgical removal of cutaneous epitheliomas and with the immediate repair of the resulting wounds by means of skin grafting suggested the application of the same method in the management of carcinoma of the larynx. Observations in using free skin grafts to correct severe cicatrical laryngeal stenosis indicated the feasibility of the procedure. The method was first applied in 2 carefully selected cases a little more than five years ago and the patients were closely observed for more than a year before further application of the plan was made. It has now been used on our service in 20 cases. During the same period thyrotomy has been performed in 293 cases and total laryngectomy in 276 cases for the removal of carcinoma of the larynx. Thus it is obvious that the procedure has only limited application. However, it must be borne in mind that the time under consideration included the period of the development of this operation and of observation of results in the initial cases; also that except for performance of the procedure all 20 of the patients mentioned above would have required a total laryngectomy. It seems certain to be carried out in a greater proportion of cases of carcinoma of the larynx in the future. In the author's experience it has been applicable in a greater number of cases than has transoral removal during the same period.

In all cases of moderately advanced malignant lesions of the larynx it has for some years been the practice of my colleagues and myself to withhold a definite decision regarding the type of operative procedure to be performed for removal pending surgical exploration. The patient is told in advance that the most conservative method consistent with safety will be followed but that total laryngectomy may be required. In most instances, exploration is made through a laryngofissure, but involvement of the anterior commissure at times renders this approach inadvisable. In this event, evaluation of the situation usually is obtained by means of transverse incisions through either or both the thyrohyoid and cricothyroid membrane. When this is not feasible because of the situation and extent of the lesion, a laryngofissure is performed and if the tumor is opened into, the margins of the laryngeal wound are thoroughly cauterized before

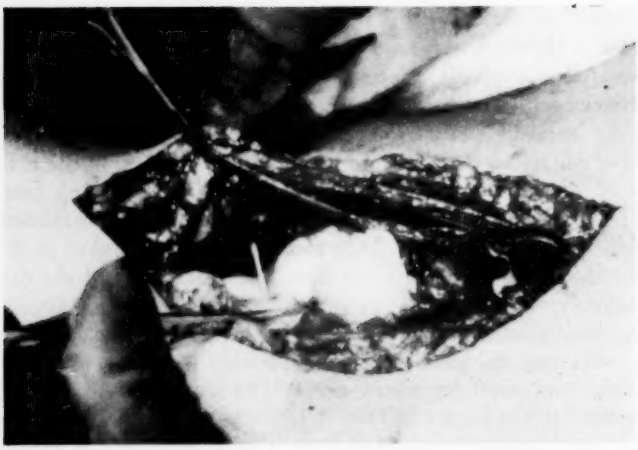


Fig. 1.—Sponge rubber stent wrapped with skin graft being anchored in larynx by means of transfixing wires.

proceeding further. As this is accomplished with regional anesthesia and the patient is conscious, he is now informed of the surgeon's recommendation and the operation is proceeded with forthright. In the past, if the neoplasm was too extensive for removal by thyrotomy or a classic hemilaryngectomy, laryngeal forceps were applied to the margins of the divided thyroid cartilage and a total laryngectomy was performed. Now, by means of hemilaryngectomy and immediate skin graft, a certain proportion of these more advanced lesions can be adequately removed without sacrificing the entire larynx, for the support furnished by the skin graft permits more extensive excision of cartilage without risk of collapse of the laryngeal lumen. Moderately advanced lesions involving the entire length of one cord, the anterior commissure and up to approximately half of the opposite cord may be removed in this manner even though fixation is present. In some of the cases in which this method was used there was in addition to the cordal growth, involvement of an entire ventricle and ventricular band. Extension onto one arytenoid was present in several instances.

Lesions of a low grade of malignancy or of moderate activity are better suited for this method of removal, as is also true of cutaneous neoplasms in which immediate plastic repair is planned since the actual extent of these slower-growing malignant tumors corresponds more nearly to their gross limits. Localized high-grade malignant tumors have at times been dealt with in this manner but because of their indefinite extent greater risk of inadequate removal is involved.

The operation consists essentially of performing a radical hemilaryngectomy, removing as much of the ala of the thyroid cartilage on the side of the principal involvement as is indicated and up to approximately one half of the ala on the opposite side, if required. Generally, a narrow strip of the superior or inferior border of the thyroid cartilage can safely be left in place and when possible this is done to lend support. One half or slightly more of the anterior or anterolateral part of the cricoid cartilage is taken away if necessary. Removal of these portions of the cricoid ring has produced no disturbances of function as would almost certainly result were the posterior portion sacrificed. The arytenoid on the involved side was removed in several of the cases but experience has shown that when this is done it is advisable to limit removal of the adjacent soft tissues as much as possible; otherwise difficulty with aspiration during swallowing may occur postoperatively. The involved soft tissues together with the overlying cartilaginous structures are excised en bloc. The trachea is opened prior to doing the hemilaryngectomy so that the respiratory tract below this level can be packed off to prevent gravitation of blood. The specimen is immediately carefully checked microscopically, using fresh frozen sections in order to make certain that there has been adequate clearance of the malignant process. Should any of the margins be too narrow, further removal is carried out. Painstaking hemostasis is essential. To secure this, the larger vessels are ligated while the smaller bleeding points are electrocoagulated.

Now, using a clean surgical setup, a dermatome graft of moderate thickness is taken from a hair-free area of the chest or abdomen, using procaine hydrochloride infiltration anesthesia. A stent of sponge rubber is prepared of sufficient size that it will exert moderate pressure upon the entire laryngeal wound when the soft tissues of the neck are closed over it. The skin graft is fastened to the stent with mastisol or rubber cement, placed in the laryngeal defect and immobilized by transfixing it with two or three stainless steel wires which pass

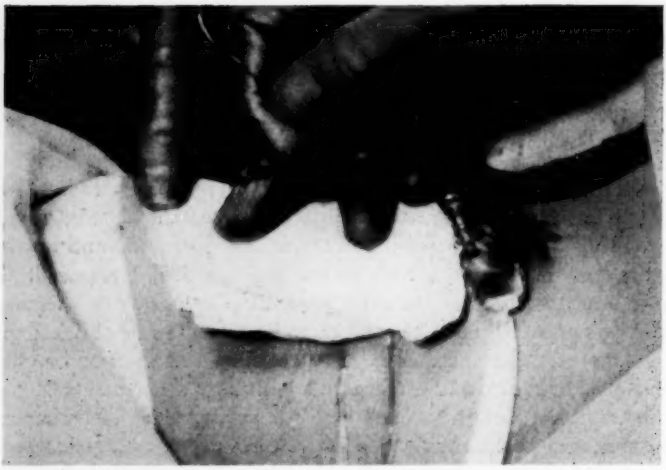


Fig. 2.—The neck wound has been closed and the transfixing wires are being tied over the gauze dressing covering it.

entirely through the soft tissues of the anterior portion of the neck and through the larynx or upper part of the trachea (Fig. 1). Additional anchorage usually is secured by passing a heavy silk suture vertically through the rubber mold and tying it to the tracheal cannula. The larynx and soft tissues of the neck are closed as in the usual thyrotomy or hemilaryngectomy. No drainage is used other than a small iodoform gauze pack below the tracheotomy tube. The ends of the transfixing wires, which have been left long, are tied firmly over a fluffed gauze pad applied to the wound on the front of the neck (Fig. 2). This is supplemented with a firm pressure bandage and the patient is urged to refrain from coughing during the first few hours.

The postoperative course generally is quite mild. Antibiotics are given routinely as a prophylactic measure and sedatives are administered as necessary for cough or discomfort. After approximately ten days, the patient is anesthetized intravenously with pentothal sodium and the larynx is exposed with the suspension apparatus. The transfixing wires and the ligature attached to the trache-

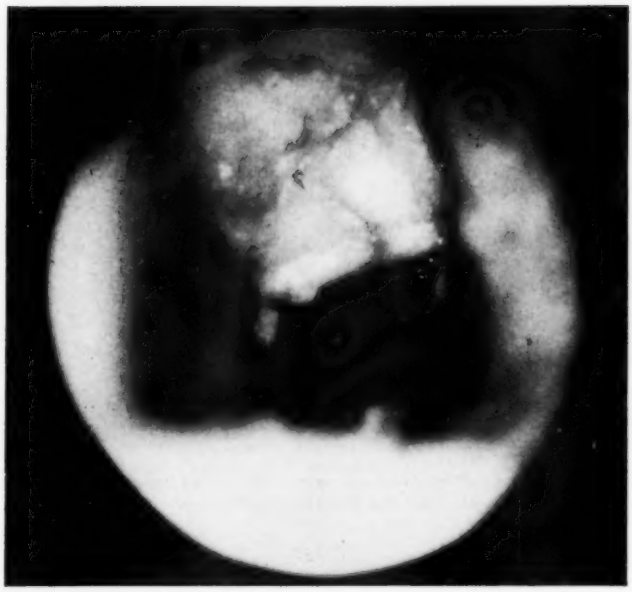


Fig. 3.—Skin graft in larynx two and a half months after hemilaryngectomy. Patients now well for approximately four years.

otomy tube are freed and the sponge rubber stent is removed through the mouth. Almost invariably the skin graft will be found to have taken perfectly where it has covered denuded areas. Elsewhere the graft will have sloughed off or is necrotic and sharply demarcated so that these portions can be pared away. An elastic dilator is now made of sponge rubber covered with a condom, inserted into the larynx and anchored in position by tying it to the tracheal cannula. This is worn for ten days to two or three weeks and is then removed through the mouth. A day or two later the tracheotomy tube is lifted out and the tracheal fistula is permitted to close. Subsequently observation of the larynx is made at intervals of two to three months for a year, thence at intervals of six months to a year.

Fig. 4.—Comparative thickness and structure of laryngeal mucosa and of adjacent skin graft nine months after application of graft (x 30).

The skin graft in the larynx remains thick, dry and rigid for a variable period following the operation and in some patients ozena results from the accumulation of mucus and crusting (Fig. 3). In the course of time these features tend to undergo spontaneous correction to a great extent, the graft becoming moist, softer and more pliable. Because of its thickness and whitish color, the graft remains permanently sharply demarcated from the adjacent mucosa; however, from a functional standpoint, it serves very satisfactorily. Microscopic sections made of the laryngeal lining nine months after operation in one of these patients strikingly show the difference in thickness of the skin and the mucous membrane (Fig. 4).

The 20 patients who underwent this procedure included 19 men and one woman, ranging in age from 34 to 65 years. They had been hoarse for periods of two months to five years. In all of those with hoarseness of more than a year's duration, there had been marked accentuation during recent months. The lesions varied in situation and extent. In all of the cases there was involvement of the glottis, ranging from the entire length of one cord to the full

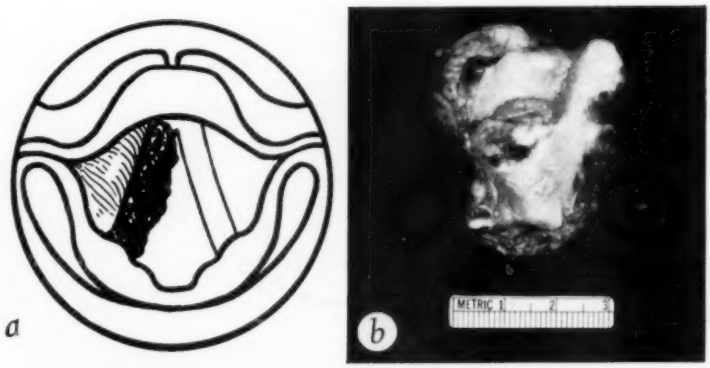


Fig. 5.—*a*. Approximate situation and extent of fixed grade 2 squamous cell epithelioma of larynx in a man 40 years of age. *b*. Specimen removed by means of hemilaryngectomy. The patient is free from symptoms and has a good, although hoarse, voice two years after operation.

extent of both cords together with the anterior commissure and one arytenoid. In addition to the cordal infiltration there was extension into the ventricle and ventricular band in most instances, also varying degrees of limitation of movement, up to complete fixation (Figs. 5 and 6). In one patient, the tumor extended form the posterior commissure over the right arytenoid and involved the entire length of the right cord and ventricle, the anterior commissure and the anterior two thirds of the left ventricule. In another there was involvement of the full length of both vocal cords, the anterior commissure and the left arytenoid without fixation (Fig. 7).

In still another patient the lesion was situated on the ventral aspect of the anterior two thirds of the right cord and the anterior half of the left cord and extended to the upper border of the second tracheal ring. This patient, as well as several others in the group, had previously been advised to undergo total laryngectomy but had refused. As a matter of fact, at the time surgical exploration of this patient's lesion was carried out, removal of the entire larynx appeared to be the only procedure that would permit adequate removal of the lesion. As he still was unwilling to accept loss of the larynx, a sub-

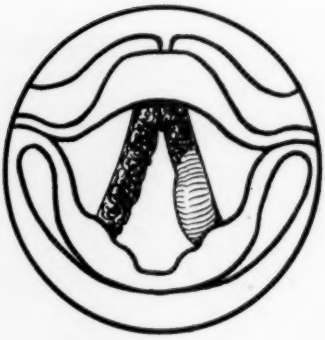
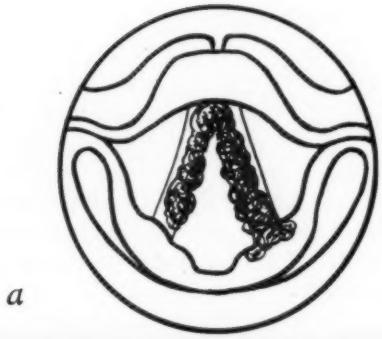


Fig. 6.—Situation and extent of a grade 1 squamous cell epithelioma of the larynx in a man 52 years of age. The posterior portion of the left cord was covered by a myxomatous lesion.

total laryngectomy was performed after the trachea had been opened, through the fifth ring. The operation required sacrifice of a portion of the right arytenoid, the entire length of the lower part of the right cord, the anterior part of the inferior aspect of the left cord, most of the cricothyroid membrane, a little more than the anterior one half of the cricoid and the anterior one half of the first two tracheal rings. A vertical bridge of subglottic mucosa, only 1 cm in width, remained below the posterior commissure. It was feared that this radical removal of laryngeal and tracheal structures probably would result in collapse of these parts and in serious disturbance of function. However, the skin graft immediately applied to line the wound has afforded ample support and at present, eighteen months after operation, there is no functional impairment and the voice is essentially normal.

The great majority of the laryngeal carcinomas in this group of patients were of moderate activity, 17 of the 20 being graded 2 on a basis of 1 to 4. Only one of the tumors was graded 1 and 2 were graded 3. One of the grade 3 lesions was considered by the pathologist to be of grade 4 at the time it was studied in fresh frozen sections made at operation. Because of the tendency for lesions of this degree of activity to infiltrate well beyond the apparent gross



a

b

Fig. 7.—Drawing and actual photograph of grade 2 squamous cell epithelioma of the larynx in a man 55 years of age. The lesion involved the entire length of both vocal cords, the anterior commissure and the left arytenoid. Patient is now free of complaints other than hoarseness two years after removal and skin graft.

limits of the disease, correspondingly wider removal was carried out and in one of the patients, a man 36 years of age, fractional roentgen therapy was given postoperatively.

The size of the specimen removed from the larynx in these patients varied considerably. This was determined largely by the extent and activity of the tumor but also in part by its situation. The fact that the lesion was removed along with the overlying portion of the thyroid cartilage and if necessary a part of the cricoid as well appreciably increased the thickness of the specimen. Several of these ranged from 4.5 to 5.5 cm in their greatest dimension. The largest measured 5.5 by 5.5 by 0.8 cm. These measurements do not allow for shrinkage following excision.

The voice in most of these patients has been somewhat hoarser than that after the usual thyrotomy. This is as one might well anticipate in view of the greater sacrifice of tissue and the resulting more limited mobility of the laryngeal structures. As following thyrotomy, the voice tends to become stronger and clearer in the course of time but only infrequently is a fair degree of resonance regained after this procedure. Generally the patients are in excellent spirits and deeply appreciative since they realize that they retain an essentially normal voice and have been spared the necessity of a permanent tracheotomy.

Experience has shown that much wider removal of tissue can be carried out safely in the anterior portion of the larynx than in the posterior part and less disturbance of function will result. Sacrifice of the entire epiglottis and one aryepiglottic fold, ventricular band and ventricle is likely to cause less difficulty with aspiration during deglutition than removal of one arytenoid. However, there is a decided variance in the difficulty experienced by different individuals following excision of corresponding tissues of the posterior part of the larynx. After limited parts of both arytenoids were taken away, no trouble with aspiration of fluids was experienced and most patients had only slight difficulty after sacrifice of an entire arytenoid. On the other hand, in one patient in whom one arytenoid was removed serious disability developed. His convalescence was protracted because of the necessity of continuing tube feeling for a number of weeks. Aspiration pneumonia ensued after the tube feeding was discontinued and eventually it became necessary to sacrifice the remainder of the larynx even though there was no recurrence of

the carcinoma. Convalescence following the laryngectomy was uneventful.

Patients who had undergone this procedure have now been followed for a few months to more than five years, three fourths of them for more than one and one-half years. To date none has presented a recurrence of the malignant tumor and except for the patient who developed aspiration pneumonia and required total laryngectomy, there have been no complications or complaints. All have resumed their original occupation.

The risk incident to the operation seems slight, although one death must be charged to the procedure. The patient was a man 62 years of age who was known to have coronary sclerosis. Death occurred suddenly following removal of the elastic dilator using suspension laryngoscopy and light intravenous pentothal sodium anesthesia and after the surgeon had left the operating room. The airway was unobstructed and although a necropsy was not obtained, death appeared to be of cardiac origin. It would seem that this termination might equally well have occurred while the patient was sitting quietly in his home or while walking along the street.

SUMMARY

A report is presented of 20 cases in which hemilaryngectomy with immediate skin graft was carried out for the removal of moderately advanced carcinoma of the larynx.

The procedure affords a highly satisfactory means of dealing with laryngeal carcinomas that are too extensive for removal by a thyrotomy or a classic hemilaryngectomy. It offers a good prognosis and does not destroy the voice or interrupt the airway.

XXXIX

TREATMENT OF HYPERPLASTIC SINUSITIS

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Hyperplastic sinusitis may be defined as a type of chronic sinus disease manifest by polypoid thickening of the nasal and sinus mucosa. In addition, one can usually see mucopus in the nose and nasopharynx and usually pus can be washed out of an antrum. I agree with Kern and Schenck[6] and use the presence of nasal polyps as one reliable index of the allergic constitution. In my opinion this disease is due to local sensitization of the submucosal tissues to bacterial proteins, most often those of the Gram positive cocci. I do not think that polyps are due to food or inhalant sensitivity.

Quoting Rich,[13] "It is important to recall that there are two main types of sensitization, which differ from each other in a number of ways. One of these is the tuberculin type of hypersensitivity, so called because the reaction of the tuberculous body to the proteins of the tubercle bacillus is its prototype, though the same form of hypersensitivity develops as a result of infection with other bacteria, fungi and filterable viruses. The other is the anaphylactic type which, too, may result from infection with microorganisms, but also from contact with a wide variety of substances derived from other sources. Each type arises from a bodily alteration resulting from parenteral contact with a foreign antigen, or with a non-antigenic substance that is capable of uniting readily with body protein to form an antigen foreign to the host. When this bodily alteration has occurred, tissues will be injured, and even killed, by local contact with amounts of the foreign substance that would be harmless to the normal body; and constitutional symptoms, and even death will occur when amounts that are entirely harmless to the normal body enter the blood stream of the hypersensitive one.

" . . . We found that while in both types of hypersensitivity extravascular tissue cells can be damaged and killed at the site of

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injection of the specific antigen, the death of the cells in anaphylactic local reactions is the result of vascular damage that interferes with the nutrition of the cells, whereas in the tuberculin type of hypersensitivity the cells of even an avascular tissue such as the cornea, or, indeed, cells from the sensitized body, washed and isolated in tissue culture, are killed by contact with the specific antigen.

"The *tuberculin type* of hypersensitivity can play an important role in the pathogenesis of infection. As a result of the development of this type of sensitization, even microorganisms which possess little toxicity for the normal body become able to damage seriously the tissues of the sensitized one."

METHOD OF DIAGNOSIS

History, of course, is important in eliciting etiology. Many patients stated that their symptoms began with an acute severe febrile respiratory infection. The usual symptoms are nasal obstruction, nasal and postnasal discharge, which is frequently yellow in color, and anosmia. It is interesting that patients with long-standing and severe hyperplastic sinusitis seldom have headache.

The usual ear, nose and throat examination is carried out and smears and cultures are made from the exudate in the middle turbinate region. I use smears stained with the Gram and Hansel methods. It is important to examine the smears under an oil immersion lens; otherwise the heavy bacterial flora usually present will not be seen. Cultures are made on blood-agar plates, including sensitivity cultures using penicillin, streptomycin, erythromycin and terramycin. Radiological examination is always necessary. In my experience the use of contrast media is seldom necessary. Puncture and lavage of all cloudy antra is, for me, an essential diagnosis method. I prefer needle puncture under the inferior turbinate and I use an 18 gauge 3½-inch straight needle because this method permits lavage of antra of patients of any age, no matter how abnormal the intranasal anatomy.

We make our own allergy studies, fully realizing that the history is more important than the skin tests. However, we do make 70 skin tests by the skin puncture method described by Walker.[11] In addition I make three intradermal tests with Endo's house dust extract, using 1:10,000, 1:100,000 and 1:1,000,000 dilutions.

Some of these patients with hyperplastic sinusitis had positive reactions to Endo's house dust extract with no clinical evidence of sensitivity to dust. My deduction was that the positive skin tests were due to bacterial proteins in the house dust extract.

The frequent association of hyperplastic sinusitis with infectious asthma proves not that one disease causes the other, but that both have a common etiology, namely sensitivity to bacterial proteins.

It should be realized that the same individual may have food or inhalant sensitivity, bacterial allergy, physical allergy and vasomotor rhinitis due to emotional turmoil. Therefore, all four factors should be looked for and treated if found.

I do examine smears for eosinophils. It is my considered opinion that the dictum that the presence of eosinophils means "allergy" has done more harm than good because it has led many people to place great stress on foods and inhalants as the probable cause of nasal symptoms so that infection as a cause of symptoms was ignored or undertreated. The fact that there are large numbers of eosinophils in the exudate as well as in the tissues of patients having bacterial allergy has been amply demonstrated by Cooke,[1] Grove[5] and Silcox.[8, 9] My experience is in complete accord with these observers.

There are, of course, other causes for eosinophilia. Stewart Wolf[12] states that nasal hyperfunction in association with stress is accompanied by a marked eosinophilia locally and in the peripheral blood as well.

SELECTION OF CASES AND DATA DERIVED

In an effort to collect reliable data for this paper, I studied the charts of 50 patients who had chronic hyperplastic sinusitis. The criteria for selecting these cases and throwing them into one group for analysis were the presence of polyps in the nose, plus density in the sinuses demonstrated by roentgenograms.

There were 23 females and 27 males, too small a sex difference to be statistically significant. Twelve patients had unilateral disease. In my opinion, the strongest evidence that hyperplestic sinusitis is due solely to bacterial allergy is presented by the *unilateral* cases, meaning those individuals who have pus and polyps only in one side of the nose. The raidological examination of these patients showed clouding of the antrum and ethmoid on the side of the polyps and

TABLE I.

AGES OF PATIENTS BY DECADES.

1 - 10 years	0 cases
11 - 20 years	1 case
21 - 30 years	7 cases
31 - 40 years	10 cases
41 - 50 years	8 cases
51 - 60 years	16 cases
61 - 70 years	8 cases

pus was washed from the antrum. Some of these individuals did, in addition, have a history of food or inhalant sensitivity and had positive skin tests, but the opposite side of the nose appeared normal and roentgenograms showed clear sinuses on the normal side.

Ten of the 50 patients in this series had asthma, usually due to an obvious chronic suppurative bronchitis, and three of the fifty had bronchiectasis. The other 37 patients had no chest symptoms, which I think is strong evidence that lymphatic drainage from the sinuses to the chest is not the reason for development of chest symptoms when it does occur. They all had lymphatics.

The duration of nasal symptoms is shown in the following table.

TABLE II.

DURATION OF SYMPTOMS.

6 - 12 months	8 cases
1 - 5 years	18 cases
6 - 10 years	7 cases
11 - 20 years	10 cases
21 - 30 years	7 cases

The symptoms most frequently listed were nasal obstruction, nasal and postnasal discharge and anosmia. Headache was seldom a symptom. Following removal of polyps and ethmoidectomy it was not unusual to find the sense of smell returning even when it had been

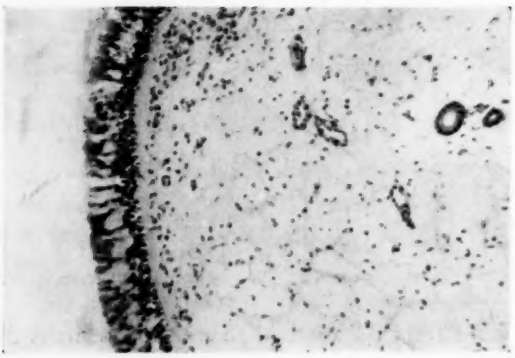

Fig. 1

absent five or ten years. One patient who had had no sense of smell for twenty-two years reported a normal sense of smell after a bilateral Caldwell-Luc operation and a bilateral transantral ethmoidectomy.

Microscopic examination of the tissue removed in cases of hyperplastic sinusitis shows a type of inflammation differing from ordinary inflammation in (1) excessive edema; (2) increased numbers of eosinophils; (3) relatively fewer neutrophils.

Eggston and Wolff[3] state: "The hypertrophic type of sinusitis may be explained histopathologically on the basis of an inflammatory reaction around the veins and lymphatics, a periphlebitis or perilymphangitis. These inflammatory processes result in obstruction of these respective vessels, causing a loss of fluid into the tissues, which distends the stroma of the tunica propria. This, in turn, is followed by various degenerative changes in the surface epithelium, glands, and bones, with distention of the tissues and polyp formation."

In my opinion the cause of the pathologic changes seen in hyperplastic sinusitis is suggested by the work of Schenck and Abell,[7] who observed the anaphylactic reaction in the living tissues of a rabbit's ear: "We concluded that the injection of horse serum into the rabbit, sensitized to this antigen, causes:

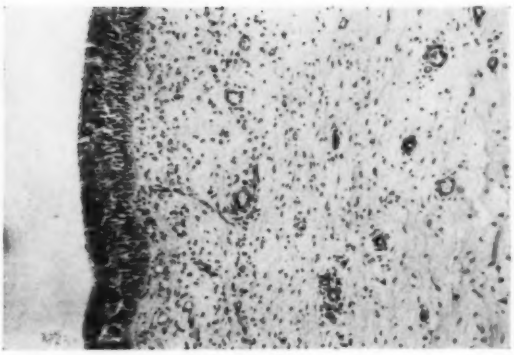

Fig. 2

1. Arteriolar contraction, which blocks the circulation;

2. A change in consistency of the endothelium, such that leukocytes adhere to the walls of the capillaries and venules in large numbers, and emigrate into the surrounding tissue;

3. A change in the leukocytes so that they become *sticky toward each other,* frequently occluding the capillaries and small venules, and sometimes forming leukocytic emboli;

4. No contraction of blood capillaries or of small venules occurs."

Thus, in my opinion, the perivascular leukocytic infiltration observed by Eggston and Wolff and seen in our own tissue specimens is explained by the *in vivo* observations of Schenck and Abell.

The histologic examination conveys the impression that in most cases these tissue changes are reversible unless there are discrete abscesses or necrosis. It was this observation that led me to try using cortisone to shrink the edematous infiltrated tissue. An additional thought was that germs are the stimulus for regrowth of such tissue. Therefore, if the germs could be eradicated by *bactericidal* doses of penicillin the tissue hypertrophies would not recur at least until an-

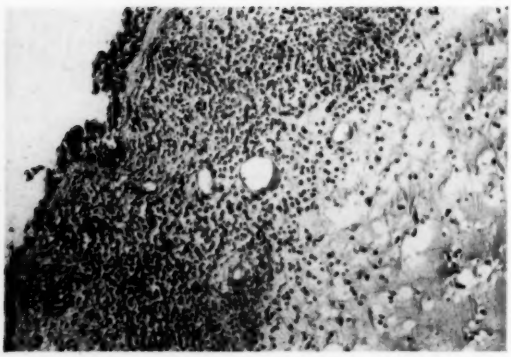

Fig. 3

other infection developed. The tissues examined were usually well vascularized, which led me to think that penicillin could diffuse effectively into the infected tissue. (See Figs. 1 and 2.)

Some type of staphylococcus was recovered in 38 cases. Twenty-seven of them were hemolytic strains and only eight were coagulase positive. Ten were not sensitive to five units of penicillin. This explains why even five million units a day may be inadequate. Eagle and associates[2] say that a serum concentration ten times the sensitivity level will usually provide the maximally effective concentration at the site of infection.

TREATMENT

In my experience there are several useful methods of treatment of hyperplastic sinusitis and usually the best results can be achieved by combining treatment methods. The methods I find useful are:

1. Antibiotics in bactericidal doses;
2. Vaccines to increase immunity;
3. Surgery to remove tissues which have suffered irreversible damage, and also to provide ventilation and drainage;
4. Management of co-existing food or inhalant sensitivity.

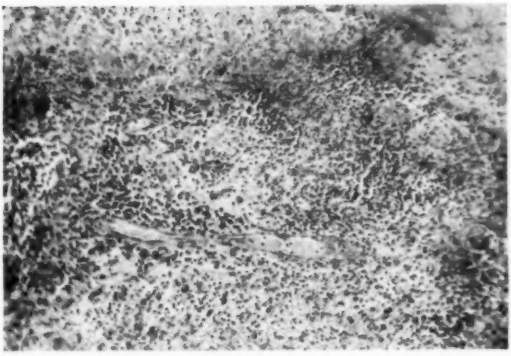

Fig. 4

In the treatment of these patients it has been my purpose to utilize the smallest amount of surgery which will produce permanent relief from symptoms. The review of these cases leaves me with the conclusion that by using 4 or 5 million units of penicillin intramuscularly daily for periods of seven to ten days, I have been able to clear up infections with intranasal surgery that otherwise would have required the external operative approach for the purpose of complete removal of the sinus linings.

The question should properly be raised: What is reversible and what is irreversible disease? Most of our tissue specimens show reversible changes; namely, marked edema of the submucosa and varying degrees of cellular infiltration, chiefly with plasma cells and eosinophils. (See Figs. 3 and 4.) The ciliated columnar epithelium is surprisingly normal in most cases. It is this histologic evidence that led me to try minor surgery such as intranasal antrostomy and removal of the pedunculated polyps, combined with bactericidal doses of penicillin, plus cortisone, to shrink the remaining hyperplastic tissue and thus afford better drainage from the ethmoids.

In my experience the antibiotic of choice is fortified duracillin, a million or more units intramuscularly every four hours for a period of at least a week, and I think my results have been better since I

TABLE III.

FIFTY CASES OF HYPERPLASTIC SINUSITIS.

Treated by removal of polyps, penicillin 4 or 5 million units I. M. daily, plus the surgery indicated. Twelve patients also had submucous resection of the septum.

	CASES	RESULT			
		EXCELLENT	GOOD	FAIR	POOR
Unilateral antrostomy	7	7			
Unilateral intranasal ethmoidectomy	1	1			
Unilateral antrostomy plus unilateral intranasal ethmoidectomy	4	4			
Bilateral antrostomy	16	11	3	1	1
Bilateral intranasal ethmoidectomy	4	4			
Bilateral antrostomy plus bilateral intranasal ethmoidectomy	14	9	3	1	1
Bilateral Caldwell-Luc plus bilateral transantral ethmoidectomy	4	4			
Total	50	40	6	2	2

There were no deaths and no post-operative complications.

have been using two million units intramuscularly every four hours. In addition, I frequently give a half gram of streptomycin intramuscularly every eight hours for 7 days. In my experience topical application of antibiotics is without value. I also think vasoconstrictors have very minor therapeutic value.

Recently I have found cortisone to be of considerable value in treatment of these patients because it suppresses the inflammatory reaction, shrinks the hyperplastic tissue and thus improves drainage.

Cortisone has great value in suppressing asthmatic symptoms, so that the necessary sinus surgery can be done. The initial daily

dose of 200 mg. by mouth was gradually reduced over a two-week period, so that the average total dose per patient was 900 mg. No toxic effects were seen in the ten patients treated with Cortone.

Cortisone is of distinct value also during the week following operation because it decreases postoperative edema. It suppresses formation of granulation tissue, but does not interfere with regeneration of epithelium. Its action is transient. It does not kill germs. Hyperplastic tissue recurs unless the germs are killed.

COMMENT

Table III shows the results of treatment. The four patients listed as having only fair or poor results should have had more complete surgery. I now think that vaccines should have been used more frequently. Only seven of these fifty patients had vaccine therapy. Its value has been demonstrated by Silcox[9] and Solis-Cohen.[10]

Fenton[4] pointed out the value of vaccine therapy in a paper given at the meeting of this Association in 1925. The fact that some of our cultures show staphylococci resistant to 20 units by the tube dilution method indicates that vaccines or radical surgery must be used in some cases.

GEISINGER MEMORIAL HOSPITAL.

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XL

SOME RECENT ITEMS OF INTEREST TO LARYNGOLOGISTS CONCERNING ANESTHESIA

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It may surprise you to hear me state that there have been few advances in anesthesia in recent years. Considerable discussion goes on concerning advances in anesthesia but, actually, are they advances? More correctly, in my opinion, these so-called advances are refinements. By and large, the same anesthetics of years ago are still in use. The methods of use have been improved; their mode of action is better understood, and certain new drugs have been added as adjuncts. Many of the complications and drawbacks of yesterday have been eliminated. The number of recently introduced refinements is too numerous for any detailed enumeration here. I, therefore, will confine my remarks to two items on anesthesia which I know will be of interest.

The first of these is the so-called hypotensive anesthesia. Actually the term hypotensive anesthesia is a misnomer. More correctly it should be called deliberately induced or intentional hypotension. Like many other supposedly new things it is a revival of something old. For some time it has been known that sympathectomized subjects tolerate blood pressures at shock level with greater ease for a longer time than those who have normal vasomotor control. The autonomic nervous system consists of a plexus of nerves and ganglia which supply the heart, blood vessels, glands and viscera. In hypotensive anesthesia the vasomotor control of the vascular system is interrupted. A blockade of vasoconstrictor impulses or an increase of vasodilator impulses cause an increase in the cross sectional area of the vascular bed and a fall in blood pressure. Increasing the total volume of the vascular compartment may be accomplished in a num-

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ber of ways. A drug which acts on the midbrain where the autonomic centers are located may be administered.

Apresoline is an example of such a drug. The number and frequency of sympathetic impulses is decreased and vasodilatation results. Its use during surgical anesthesia is not practical, however, and other methods must be used.

One may cause vasodilatation by using drugs which affect the centers in the medulla. The vasomotor center may be depressed. Narcotic and hypnotic drugs accomplish this to a certain extent. Barbiturates and morphine depress the center and a fall in blood pressure results. The disadvantage in using these is that other centers, notably the respiratory centers, are depressed along with the vasomotor center.

A drug which stimulates the vasodilator center in the medulla may be used also. The veratrum alkaloids behave in this manner. Likewise they are not practical for use during surgery as they lack controllability. The preganglionic fibers as they emerge from the spinal cord may be blocked and the blood vessels then become relaxed. This may be accomplished by injecting procaine into the subarachnoid space. This procedure, known as total spinal block has been used to a large extent by the British. Its chief drawback is its lack of controllability. Peridural blocks act in the same manner but are somewhat more difficult to execute technically. The ganglia may be blocked directly with procaine but this is somewhat tedious and not effective in securing the generalized vasodilation which is required.

Ganglionic blockade is more effectively obtained by the use of systemic acting drugs. The ganglionic blocking agents available for this purpose are quite effective. Hexamethonium, Etamon, Arfonad and Pendiomide are the currently employed drugs. This is the most extensively used and practical method of inducing hypotension during surgery. Arfonad and Pendiomide act briefly. They thus permit greater control of the pressure and prompt reversal of the vasodilatation when it is desired to interrupt it.

If desired, the same effect may be obtained by causing a blockade at the nerve endings by means of a sympatholytic drug. Dibenamine, Priscol and Benodaine are examples of drugs which cause a blockade at this site. These drugs are used to a lesser extent than the ganglionic blocking agents.

One may go beyond the nerve endings and administer a drug which depresses the smooth muscle of the blood vessels directly. The nitrates and nitrites are used for this purpose. They are not practical for hypotensive anesthesia because they lack controllability. The pressure may also be lowered by decreasing the blood volume by employing the procedure known as arteriotomy. A cannula is placed in the radial artery and blood is withdrawn temporarily into a container with an anticoagulant. It is reintroduced when the need for the hypotension is over. The chief disadvantage of this method is that generalized vasodilatation is not present and vasomotor control remains active. In due time irreversible shock may occur. Also it is tedious, cumbersome and involves sacrifice of an artery.

The chief use of controlled hypotension is for situations in which considerable bleeding is anticipated. The hypotension is induced during surgery just prior to the moment that bleeding is anticipated. A relatively bloodless field results for a brief period and the surgery can be accomplished with greater ease. Controlled hypotension facilitates neurosurgery when highly vascular tumors or aneurysm are to be excised. It facilitates surgery about the head and face since the blood vessels are abundant in these areas. The technique may be applicable to radical dissections of the neck, resections of the mandible, laryngectomies and other operations in which excessive blood loss is anticipated.

Obviously there are bound to be objections and hazards in the use of this method and it cannot be employed routinely in every case. The relaxation of the vascular bed, of course, reduces the blood flow through the vital organs. If the cardiovascular system is diseased the incidence of complications, untoward reactions and undesirable sequela mounts. The circulation time is prolonged, sometimes twice the normal value. When the coronary arteries are diseased, the myocardium may not receive an adequate blood supply and coronary insufficiency follows. Coronary thrombosis is a likely occurrence. The brain likewise may suffer damage to lack of nourishment or oxygen deficiency. The slowing of the circulatory system in sclerotic vessels predisposes to thrombosis and later emboli in any organ. Cerebral thrombosis and coronary thrombosis have been the most frequent complications of the technique. At least 60 mm of mercury pressure is necessary to maintain adequate filtration by the kidney. If the blood pressure remains below this level for any length

of time, damage to the tubules may result. Anuria follows in the post-operative period.

In executing the technique the patient is anesthetized in the usual manner. Ten or fifteen minutes before the anticipated time of excessive bleeding the vasodilator drug is administered intravenously. Usually the blood pressure is reduced to 60 to 80 mm of mercury. If maintained above 80 mm of mercury, the results are not satisfactory. The blood in the vascular space is influenced by gravity and may be shifted about at will. The pressure may be reduced further by inclining the patient with the head up and the feet down. If the pressure falls below the desired depth, the head may be tilted downward and the feet up. The effects of gravity cause an increase in the amount of blood returned to the heart and brain and the pressure rises. After the denervation is instituted the blood pressure is controlled by varying the position of the patient until the effects of the drug wear off. Blood must be replaced as it is lost. These patients do not tolerate blood loss. Little anesthetic is required during the period of hypotension. The depth of anesthesia necessary is nowhere as much as it is without hypotension. The period of hypotension should be as brief as possible, not to exceed one hour at the most. At the conclusion of the portion of the operation requiring the hypotension the blood pressure may be raised by tilting the patient or by injecting a vasoconstrictor drug such as Neosynephrin, Arterenol or Desoxyephedrine.

The most frequent complication in the post-operative period is slow recovery. Patients require a long time to recover from the post-anesthetic depression. The second frequent complication is reactionary hemorrhage. Bleeding points which have not been detected and tied off before closure resume bleeding as the pressure rises. Anuria, cerebral thrombosis, apoplexy, coronary thrombosis, pulmonary embolus, encephalopathy are occasional complications encountered.

The technique is not to be used routinely. In the light of our present knowledge its use in benign conditions is not justified except in circumstances in which the possibility of serious sequela is the lesser of two evil choices. One cannot deny that it does facilitate the more formidible types of surgery and if used with discretion in malignant conditions, is a worthwhile procedure notwithstanding the possible complications. The procedure is not an anesthetic one. The anesthetist merely executes the technique because he ordinarily

watches the blood pressure for the surgeon and is in the best position to direct its management. The procedure is merely one of disturbing the physiology during surgery in order to accomplish vasodilatation.

The second item of interest to surgeons is the use of muscle relaxants as adjuncts to anesthesia. In laryngology the muscle relaxants play an important role, particularly for endoscopy. Originally, the sole muscle relaxant available was curare. Before 1936 specimens of curare were of doubtful purity. The drug was nothing more than a pharmacological curiosity. Richard Gill, an American explorer was instrumental in locating and bringing to the United States from South America the authenticated botanical specimen of curare. The active principle in curare is the alkaloid tubocurarine. Since the reintroduction of curare and isolation of its active principle many other synthetic muscle relaxing agents have been prepared. Each of these has certain merits and demerits. The majority of these have many features in common and it is best to classify and study them in groups rather than as individual compounds. From a pharmacologic standpoint these compounds fall into major groups. The first group, which includes curare, its alkaloid, tubocurarine and synthetic substances derived from the alkaloid, such as methyl tubocurarine, interfere with the action of acetylcholine on the somatic nerve endings. It is now recognized that as the impulse in a somatic nerve fiber supplying skeletal muscle, reaches the nerve ending, acetylcholine passes through the subneural space to the neuro-junctional membrane. This membrane is polarized in its resting state. As the acetylcholine reaches the membrane depolarization occurs and contraction of the muscle fiber is initiated. As soon as the stimulus resulting from the depolarization recedes the muscle relaxes to its resting state and remains relaxed unless the membrane becomes repolarized so that it will be influenced by the next impulse. In the meantime, cholinesterase splits the acetylcholine into acetic acid and choline which are physiologically inactive. Removal of the acetylcholine causes the membrane to regain its polarity and is then ready to receive the next impulse. The next impulse once again liberates more acetylcholine and the cycle is repeated. When tubocurarine and substances which act similarly are administered, it fixes itself in some manner at the membrane and prevents the acetylcholine from causing depolarization. The membrane then remains polarized and the muscle fiber is unable to contract even though acetylcholine is liberated with each impulse coming down the nerve fiber.

Curare, tubocurarine, metubine, flaxedil and mytolon all act in this manner. The blockade caused by these drugs may be reversed in one of two ways. Substances which inhibit cholinesterase cause acetylcholine to accumulate at the membrane in sufficient concentration so that it then is able to act on the membrane. Eserine and prostigmine both inhibit cholinesterase from destroying acetylcholine. Theoretically they reverse the action of curare and curare like drugs. In actual practice, however, the results are disappointing. A recently introduced substance related to prostigmine known as tensilon is an effective antagonist. Presumably it acts by combining with the relaxant or removing it from the site of action at the myoneural junction. Acetylcholine then is free to initiate the contraction in a normal fashion.

The second group of muscle relaxants act in a somewhat different manner but all in the group are pharmacologically similar. They cause relaxation by causing persistant depolarization. If acetylcholine is present in excessive amounts and remains at the junctional membrane, the membrane remains depolarized. The muscle fiber then remains relaxed. Any drug which has an acetlycholine action and causes sustained depolarization of the junctional membrane will produce relaxation. A contraction preceeds the relaxation. When administered to a patient such a drug induces fasciculations for 20 to 30 seconds, then relaxation follows. Syncurine is the better known drug of this type. A newer drug which offers promise of being clinically useful is succinyl choline (Anectine). It appears to be safer than the muscle relaxants heretofore studied. This is largely due to the method by which it is detoxified. Most muscle relaxing agents are either eliminated unchanged by the kidney or are destroyed by the liver or by a combination of both methods. Their duration of action varies depending on the rate of elimination. Some act longer than others. Succinyl choline, however, is different in this respect. Succinyl choline is an ester. Like acetylcholine it is attacked by the naturally occurring enzyme, cholinesterase, which converts it into succinic acid and choline. Both of these products of detoxification are naturally occurring body constituents. It is advantageous to have the detoxification products of a drug not be biologically foreign to the body. The destruction occurs rapidly. Usually its effects have disappeared within two to three minutes.

There are several other reasons why succinyl choline is more desirable than other muscle relaxants. In many instances when

muscle relaxing agents are used as adjuncts to anesthesia, the respiratory pattern is disturbed and does not return to normal for quite sometime. Succinyl choline, however, does not appear to act in this manner. As soon as the paresis has disappeared the respiratory pattern is restored to normal. Then, too, when succinyl choline is used one may interrupt the blockade at any time desired. This cannot be done with the other muscle relaxants. If a sustained blockade is desired for many minutes, the drug may be administered by a continuous intravenous drip. It must be remembered that neither prostigmine or tensilon are effective for reversal of the action of syncurine and succinyl choline. As a matter of fact, the prostigmine may enhance and prolong the effect. No suitable antagonist has been discovered for these two agents. The doses ordinarily used for muscle relaxation do not appear to affect the central nervous system. There is neither stimulation nor depression.

It must be remembered that muscle relaxants are not anesthetic substances. An operation may be performed upon a subject using a muscle relaxant alone, if one chooses to do so. However, the operation is performed without any resistance because the subject is incapable of voluntary movement. The central nervous system is still active. The subject feels the pain and remembers the experience but is unable to protest. It is customary to combine the muscle relaxants with a basal narcotic, such as thiopental (Pentothal) or other barbiturate. The narcotic produces the desired hypnotic effect but still does not cause a blockade of the painful impulses. A third substance must be added for satisfactory results. The third subtsance must be analgesic or anesthetic. For this reason it is common in present day practice to use a combination of nitrous oxide which supplies the analgesia, thiopental which supplies the depression of the central nervous system which is only obtained with difficulty with nitrous oxide, and the muscle relaxant which supplies the relaxation which neither the nitrous oxide or thiopental is capable of producing. For endoscopic work the combination of a muscle relaxation and ultrashort-acting barbiturate is widely used. It must be emphasized that satisfactory topical blockade is most desirable in this type of instrumentation, otherwise excessive amounts of these drugs will be required. The difficulties which arise from these combinations in endoscopic work are largely the result of overdosage.

One must remember, too, that the muscle relaxants have other [illegible] besides those of interrupting the impulses at the nerve endings

of skeletal muscle. They also have autonomic effects, that is, they either stimulate or depress the ganglia. Large doses of curare, for example, block the autonomic ganglia. The doses that are ordinarily used during anesthesia, however, have little effect on the autonomic nervous system. Some of the newer synthetic substances have vagal stimulating effects or vagal depressing effects. The curare preparations as a whole have a histamine-like action. Occasionally bronchial constriction is encountered. This must be borne in mind during bronchoscopy and laryngoscopy. Such bronchial constriction is best relieved by the use of a bronchial muscle relaxant such as aminophylline.

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XLI

PHARYNGEAL AND LARYNGEAL PHENOMENA OF VASCULAR ORIGIN

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In the practice of otolaryngology a significant number of patients are encountered, complaining of vague pains, and various types of discomfort in different parts of the head and neck which cannot be definitely classified and for which no pathology is demonstrable to account for the cause of symptoms.

In the reported observations of Greenfield Sluder,[1] a great variety of these cases was described in which the cause was not infrequently attributed to hyperplastic sinusitis, or to involvement of the nasal ganglion and the Vidian nerve. In many instances, symptoms were referred to the pharynx or the larynx.

During the course of observation of a large number of headache patients during the past 12 years, we[2, 3] noted that not infrequently cramping pains were also described in the leg on one side or the other and that upon relief of the headache symptoms, the leg pains also disappeared. This observation suggested therefore that the arteries of practically any part of the body may become involved with the production of vasoconstrictor and vasodilator phenomena. The so-called "abdominal migraine" described by Osler certainly is of vascular origin. It is not unreasonable to assume also that in certain cases of precordial, back or pelvic pain the same type of mechanism may be concerned. We have noted that aside from the true headache, pains and discomforts may also be referred to the ear, the parotid, the maxillary and dental regions and that they may be explained on a vascular basis. Later, it was further observed that patients with painful and annoying symptoms referred to the nasopharynx, tonsillar, pharyngeal, laryngeal and deep cervical regions could also be classified as of vascular origin.

It was also observed that in some cases of headache, certain symptoms may be caused by the involvement of those vessels which

affect specific functional structures. For example, a patient with histaminic cephalgia had transitory attacks of blindness lasting 20 to 30 minutes on the side affected by the headache. The blindness, however, never occurred with the headache. Both conditions disappeared completely with histamine therapy. In another patient, swelling of the common carotid artery with marked tenderness on palpation occurred with attacks of headache involving the temporal area on one side or the other, mostly on the right. During a period of eight years of headaches, she had three attacks of Bell's palsy, one on the left, two on the right. Under these circumstances, it is reasonable to assume that the attacks of facial paralysis were of vascular origin.

In the case of another patient who is now 65 years of age, attacks of transitory aphasia occurred off and on from childhood. A sister had similar attacks. The aphasia, characterized by an inability to express himself, would last usually 1 to 3 hours, sometimes longer. Attacks would come in cycles or there would be long periods of several years with no symptoms, following the pattern of histaminic cephalgia rather than migraine. These attacks of aphasia could be controlled with the use of histamine or with the administration of ergot compounds.

We have also encountered many instances of vestibular and cochlear phenomena, which appeared to fit into these patterns of vascular involvement.

The diagnosis of these various phenomena as of vascular origin was based upon the fact that the complaints were referred to the area of distribution of certain arteries such as the lingual, the tonsillar, the ascending or descending pharyngeal, the vertebral, the thyroid, the superior or inferior laryngeal. The symptoms were comparable to these noted in cases of headache of the migraine, tension or histaminic cephalgia types, as manifested by vasoconstrictor, vasodilator and secretory phenomena. The onset of symptoms may be slow as in migraine and especially tension headache in which vasoconstriction precedes vasodilatation, or suddenly as in histaminic cephalgia. Secretory phenomena occur typically in the latter group of cases and are manifested by sweating of the skin, lacrimation, nasal discharge and stuffiness on the involved side.

In the pharynx and larynx, symptoms may be comparable to those of migraine and tension headache. With the latter, the onset

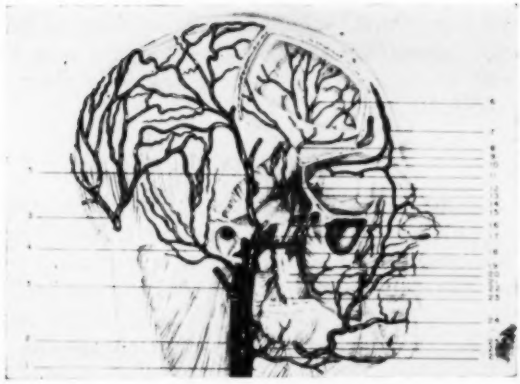

Fig. 1.—1, External carotid A. 2, Internal carotid A. 3, Occipital A. 4, Posterior auricular A. 5, Superficial temporal A. 6, Cerebral A. (Asc. Br.). 7, Frontal A. 8, Middle meningeal A. (Ant. Br.). 9, Supraorbital A. 10, Dorsalis nasi A. 11, Ophthalmic A. 12, Zygomaticoorbital A. 13, Optic nerve. 14, Angular A. 15, Infraorbital A. 16, Deep temporal A. 17, Middle meningeal A. 18, Masseter A. 19, Buccinator A. 20, Lingual nerve. 21, Inferior alveolar nerve. 22, Superior labial A. 23, Inferior alveolar A. 24, Inferior labial A. 25, External maxillary A. 26, Lingual A. 27, Facial nerve.

is characterized by premonitory visual symptoms, nausea, vomiting and a sense of tension. In the pharynx, vasoconstrictor symptoms may be manifested by a sense of constriction or choking and dryness. Upon the appearance of vasodilation, pain of a dull, throbbing or boring character may ensue. The involved vessels may be tender to palpation. As in the case of migraine, symptoms may last for 24 to 72 hours.

In the vasodilator types, following the pattern of histaminic cephalgia, the onset is sudden with symptoms of deep-seated boring pain or aching which ends rather suddenly after a few hours. Attacks are not uncommon at night, awakening the patient from sleep. These patients differ from the ther types in that there are no nervous or tension factors. The vasodilator type is a calm, relaxed individual.

DIAGNOSIS

A tentative diagnosis of these vascular phenomena is based upon the nature of the symptoms and the distribution of the pain or dis-

comfort. Final proof, however, is corroborated by the response and relief of symptoms with vasodilator agents such as histamine or nicotinic acid. Symptomatic relief of symptoms may be obtained by the use of various vasoconstrictors, especially those containing ergot compounds.

DIFFERENTIAL DIAGNOSIS

These disturbances of the vascular mechanism are not associated with any gross pathologic changes. When some pathologic condition is present, however, it must be determined whether it is or is not the cause of the symptoms. Certainly, there is a great variety of conditions which may involve the head and neck which may produce symptoms similar to those caused by vascular phenomena. There are some conditions which are readily recognized so there should be no difficulty in establishing a definite diagnosis. The most important to be considered in differential diagnoses may be enumerated as follows:

1. Edema of the uvula, palate, epiglottic, pharynx and larynx.
2. Tumors and ulcerations.
3. Tonsillar diseases.
4. Elongated styloid process.
5. Cysts, glands and tumors in the neck.
6. Thyroiditis, substernal goitre.
7. Neuritis or neuralgia of the glossopharyngeal or laryngeal nerves.
8. Ear diseases—referred.
9. Jugular-foramen syndrome.
10. Bulbar syndrome.
11. Esophageal disease.
12. Referred-cardiac.
13. Effect of drugs.
14. Effect of nose drops.

TREATMENT

Acute, more or less severe attacks may be relieved with Cafergot, D.H.E. 45 (dihydro-ergotamine), Valoctin, or hydergine. For the

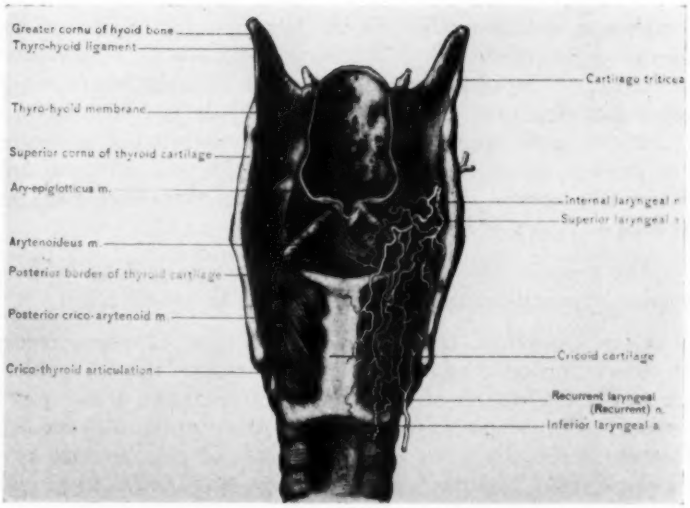

Fig. 2.—The nerves and arteries of the larynx (from Deaver).

more prolonged attacks, Bellergal may be administered 3 to 4 times daily.

The active treatment of these patients by the administration of histamine and sometimes nicotinic acid is usually followed by prompt relief of symptoms. As in the case of headache, the cycle of attacks may be broken with only a few injections. In some instances, however, prolonged treatment may be necessary. Periods of freedom from symptoms vary from months to several years.

Any procedure which increases the local blood supply, such as tonsillectomy for example, may result in a dramatic relief of symptoms, but after a period of a few weeks, the symptoms usually return. In some instances, however, the interruption of the cycle of attacks may last for six months or longer.

The following case reports present convincing evidence that these phenomena are vascular in nature:

CASE 1.—(No. 6868) J. J., a male, aged 56 years was observed for the first time on February 12, 1946. He complained of a sense of tightness and discomfort in the left laryngeal region of three months' duration. At times, there was also a sense of soreness in the tongue. The attacks would come on at night after going to sleep. Activity gave relief. Treatment with histamine (No. 6) 1-1,000,000, gave complete relief for nine months when he returned with soreness referred to the tongue. Again, treatment was effective for five months, then 4 months. For five years since then, there has been no return of symptoms.

The vascular involvement in this case appeared to be in the superior laryngeal artery and in the lingual artery (Fig. 1).

CASE 2.—(No. 11,535) Mrs. G. K., aged 58 years, reported for observation on September 5, 1951, complaining of a tender or sore area in the left side of the upper laryngeal region of many years' duration. The symptoms would recur without cause and were better when she was active. She also had attacks of pain referred to the left cheek area. Treatment with histamine (1-10,000,000) produced almost immediate relief, and after four weeks there were no more symptoms for a period of five months.

In this case, the symptoms were referred to the left superior laryngeal artery as in the preceding case. Mild involvement also occurred in the left superior maxillary artery.

CASE 3.—(No. 11,581) Mrs. M. B., aged 70 years, reported for observation on September 28, 1951. She stated that for about 25 years, she had suffered with attacks of "biting pains" first on one, then the other side of the neck, but mostly on the left, referred to the upper laryngeal region. Attacks would sometimes occur at night and awaken her from sleep. She was better when out of bed. She had never been free of attacks for longer than 1 month in 25 years. Within two weeks the symptoms were entirely relieved with histamine therapy (1-10,000,000) and there was no return after three months.

Although this case is similar to the two preceding, this patient had almost continuous discomfort for 25 years. She had many examinations and treatments with no relief.

CASE 4.—(No. 12,089) Mrs. L. P., aged 31, was seen on August 4, 1952, complaining of an intermittent burning pain in the left

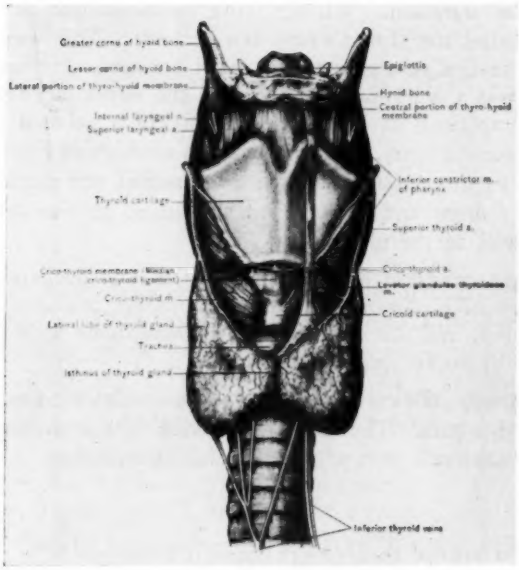

Fig. 3.—The superior thyroid artery and its branches (from Deaver).

side of the neck of three years' duration. Eight months previously, he had had a tosillectomy as well as removal of part of the lingual tonsil. For three weeks following this, there was complete relief of pain, but subsequently, the symptoms became more pronounced. All examinations were negative including a G.I. series of x-rays. With histamine therapy, supplemented with the administration of Bellergal for symptomatic relief as indicated, the symptoms were entirely relieved except for occasional attacks. Initial therapy was given for three weeks, then discontinued for three months and resumed again for three weeks. After four months, there were no significant complaints.

In this case, the pain was referred to the region of the superior thyroid artery (Fig. 2). As in most cases of this type, complete relief was obtained for about three weeks following tonsillectomy.

Case 5.—(No. 12,310) C. W., aged 47 was observed for the first time on December 29, 1952, complaining of a sensation of some-

thing sticking in the throat and a constant desire to clear the throat, of ten years' duration. While talking to the patient, it was noted that he cleared the throat every few seconds. This was very embarrassing to him in conducting a retail business. He had no pain, but there was a sensation of a lump in the upper laryngeal region. He also complained of a stuffy nose for which he used nose drops off and on for ten years. Abstinence from smoking of cigarettes made no change in the symptoms. The examination was negative except for slight redness and some overdevelopment of the veins on the posterior wall of the pharynx.

Treatment consisted of the avoidance of nose drops and the administration of histamine and Bellergal. After ten days, there was marked relief, and after six weeks, the symptoms were completely relieved with no further treatment.

In this case, the excessive use of vasoconstrictor nose drops probably played a part. The constant clearing of the throat may have been habitual but a part of the vascular disturbance.

SUMMARY

On the basis of these observations, it is reasonable to assume that certain symptoms, at times somewhat vague, referred to the arterial regions of the pharynx and larynx may be of a vascular nature. Most of these patients, because of the absence of any visible pathological changes have been classified as of the so-called, "globus" type.

In general, the symptomatic patterns resemble those of migraine, tension and histaminic headaches. Response to the administration of histamine and symptomatic relief with compounds of ergot indicate that in certain patients pharyngeal and laryngeal symptoms may be of vascular origin.

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XLII

PRECISION BRONCHOLOGY: A "MUST" FOR THE THORACIC SURGEON

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Precision is the sum total of many small details each as nearly perfect as possible, none very important by itself, but all necessary to make up a really precise whole.

For many years the author has been privileged to work closely with thoracic surgeons having a wide experience in the management of all types of pulmonary disease, serving as bronchologist and carrying out pulmonary studies prior to lung surgery. These thoracic surgeons have said many times that the bronchologist could serve them best when he learned to "think like a thoracic surgeon." As time has gone on, the precision of these bronchologic efforts has increased.

What factors distinguish a precise bronchologic study from a mediocre one? As a beginning, consider what is meant by precision. The dictionary defines it as "sharply or exactly limited as to meaning; exact, definite, not loose, vague or equivocal." This definition standardizes the word "precision" but not with respect to any particular usage. In actual practice "precision" may mean almost anything. Its meaning varies with the development of the field to which it is applied. The higher the development, the more minute the detail connoted by the term "precise." The following examples from Collier[1] illustrate this idea.

Early in the nineteenth century a machinist advertised possession of a boring mill "capable of turning the cylinders of steam engines accurate to plus or minus the thickness of a shilling." A shilling is three thirty-seconds of an inch thick. In 1953 steam engine cylinders made to an accuracy of only three thirty-seconds

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of an inch would be extremely crude. Today's tolerances would be within a few thousandths of an inch.

As a second illustration consider the problems of the highway construction engineer. A highway construction engineer could maintain the straightness of a road to plus or minus one foot per mile but this would probably be unnecessary. Plus or minus one yard per mile would probably be accurate enough. But in an international boundary survey one foot per mile would be relatively poor accuracy. What constitutes precision in one field is not precision in another; and what is precision at one stage of the development of an art or craft is not precision at a later stage of the development of that art or craft.

What can the thoracic surgeon expect from the bronchologist? The thoracic surgeon can expect that the bronchologist will be intensely interested in his work; that he will study diligently to perfect his knowledge of pulmonary anatomy, physiology and pathology; that he will fully merit being called a bronchologist or student of the bronchi rather than a bronchoscopist who sees the bronchi but may not understand what he sees. The papers of Jackson and Huber,[9] of Boyden et al,[10] and of Kane[11] will be essential to this study.

The thoracic surgeon can expect the bronchologist's description of the entire respiratory system to be of a quality to enable the thoracic surgeon to picture the respiratory system in his mind's eye just as if he himself had made the examination. The thoracic surgeon should reasonably expect the bronchologist to study the patient's problem before the bronchoscopy. The surgeon and bronchologist should discuss the entire problem, examine the x-rays and the bronchologist should talk to the patient about the bronchoscopic procedure. A few minutes of explanation prepare the patient for bronscopy. By reducing nervous tension one greatly increases the quality of the studies.

The thoracic surgeon can expect to learn the condition of the patient's pharynx and larynx, including motility and structural state of the vocal cords. He can expect to know the position, size and shape of the trachea with a description of any abnormality produced by extrinsic pressure or by intrinsic tumors or ulcers. He can expect to know the state of the mucous membrane as concerns

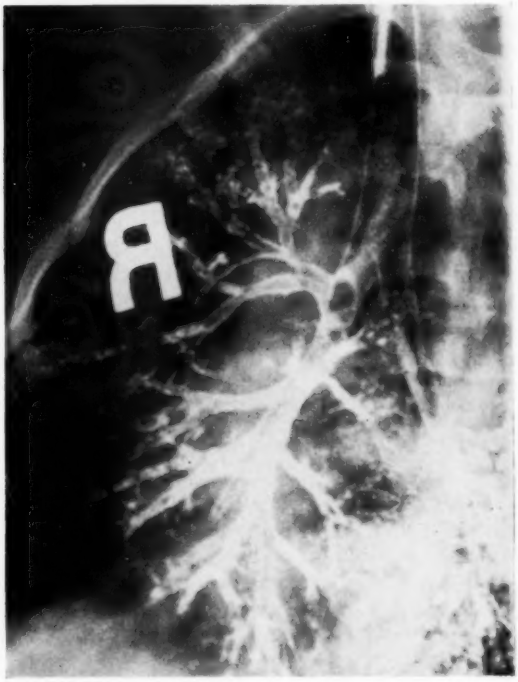

Fig. 1.—Shows normal right bronchial tree in a five year old girl who had bronchiectasis in the left lung. Note that all ten major bronchi are clearly visible plus a duplication of the apical division of the lower lobe. This is a so-called "B*" bronchus of Boyden. This bronchogram and the other children's bronchograms were obtained by the method reported by Priest.[7]

inflammatory reactions within it and evidence of suppuration as shown by pus coating the mucosa of the trachea.

The thoracic surgeon can expect a bronchologist to indicate to him what the tracheal carina looks like. It may be straight and slim covered with normal mucosa; it may be broadened due to the patient's habitus or to tumor masses lying in the angle between the right and left main bronchi. It may be kinked during the expiratory phase of respiration, and straight during the inspiratory phase

suggesting the presence of tumor masses adjacent to the carina. It may be attached to the large vessels by infiltration of tumor and therefore make excursions with the vessels in a transverse direction. These excursions will be out of phase with the respiratory excursions.

The thoracic surgeon can reasonably expect the bronchologist to be thoroughly familiar with bronchial anatomy and to examine the orifices of the ten major branch bronchi present within the right and left bronchial trees. Such examination should include routine telescopic observations of the upper lobe bronchi with the right angled telescope and occasionally with the retrograde telescope. Examination of the middle and lower lobe bronchi should include observation with the foroblique telescope except for the apical division of the lower lobes; this bronchus should be examined with the right angled telescope. Right angle and foroblique telescopes should be a normal part of the setup for each case and should not be used only in cases presenting special problems. The same observation described in connection with the trachea should be made routinely of the mucosa of the bronchi of both bronchial trees.

The collection of specimens by aspiration as described by Clerf should be routine. Material should be collected from whatever branch bronchus is indicated by previous study of the chest x-rays and by study of the patient before bronchoscopy, as well as by bronchoscopic findings. Specimens removed with the aspirating device should be subjected to certain routine tests. These should include cytologic study of the secretions for tumor cells by a clinical pathologist familiar with such study. The use of the Papanicolaou technique has been found to be extremely valuable but it is subject to limitations based on the experience of the pathologist. Early in the author's experience false positives were obtained and some patients had portions of lungs removed when it is now believed that carcinoma was not actually present. Inflammatory metaplasia of cells in lesions such as bronchiectasis is the most frequent cause of this difficulty. A second routine test which should be applied to all specimens removed by aspiration is culture for tubercle bacilli. All specimens should be labeled immediately as to their exact source within the bronchial tree. If possible the exact bronchus from which they were aspirated should be listed. It is preferable to say that one has aspirated the medial basal bronchus of the right lower lobe rather than that one has aspirated the right lower lobe bronchus. Sometimes such accuracy is not

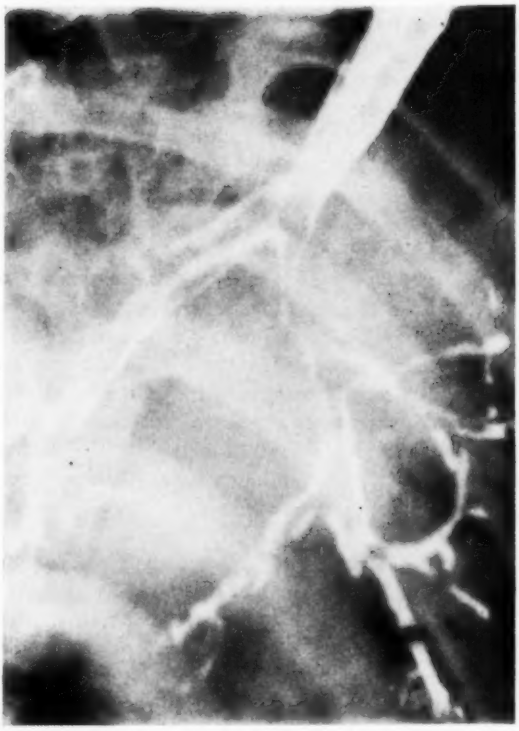

Fig. 2.—Patient was a ten month old baby who had marked whistling sound to his breathing. Bronchoscopy showed compression of left main bronchus and bronchogram shows compression below the takeoff of the left main bronchus from the trachea. The left lower lobe bronchus and the left upper lobe bronchus together with several divisions may be seen. The compression of the left main bronchus as it skirts the mass is visible. A partial bronchogram of the right bronchial tree is shown, but no effort was made to make a good bronchogram on this side.

necessary or possible but if one strives for a high level of routine accuracy he is more apt to get information of value to the thoracic surgeon than if he strives for a medium level of accuracy.

When it is possible to make a biopsy of a tumor using bronchoscopic forceps, what does the thoracic surgeon want to know about the bronchi? He wants to know how much clearance he has between the end of the tumor and the point where he will have to resect in order to achieve a cure. If the tumor is so situated that it would seem that he has ample clearance he wants to know if the bronchus proximal to the tumor is rigid suggesting tumor infiltration within or adjacent to its wall. He also wants to know if the bronchus is narrowed as though compressed by masses lying outside of it. On several occasions it has been necessary to subject patients to a second bronchoscopy because the first bronchoscopist was not sufficiently accurate in his localization of the tumor although he had obtained a positive biopsy for carcinoma. In these cases the thoracic surgeon could not tell whether there was room to attempt a resection or not. It is useful to make aspiration studies on all cases in which forceps biopsies are obtained. This gives the pathologist known positive material on which to practice his cytology. When excessive bleeding follows biopsy this is not possible.

The question of measurements within the bronchi has been raised by several authors. A bronchial yardstick and an intrabronchial caliper have been devised by Emerson.[2, 3] An experimental recording bronchial caliper has been used by Andrews.[4] The presence of such instruments on a bronchoscopic table is of unquestionable value. On the other hand, one must not forget that he can make use of simple instruments always present and can achieve reasonable accuracy of measurement sufficient for most purposes. This is especially true of longitudinal measurements of the bronchi and is not so true of measurement of bronchial diameters. One can use an ordinary suction tip and ruler. The suction tip can be placed against the tumor and the measurement made. The suction tip can then be placed against the nearest anatomic landmark, for example the tracheal carina, and a second measurement can be made. This simple method gives excellent results and removes from the thoracic surgeon's mind any doubt as to the accuracy of the bronchologist's observation. Such statements as "a long way down the left main bronchus," "half way down the left main bronchus," "it was a preety

Fig. 3.—Shows bronchogram of a seven year old girl. There is marked cylindrical bronchiectasis of all branches of the left lower lobe while the upper and lower divisions of the left upper lobe are shown to be entirely free from bronchiectasis.

big one," and so forth have no place in the accurate bronchology of today. They may have been adequate in that phase of bronchology corresponding to the three shilling tolerance of the steam engine but they are not sufficiently accurate for today.

The preparation of proper bronchographic studies using contrast medium injected into the tracheobronchial tree has been discussed by many authors and will be touched upon briefly here. As in other phases of precise bronchology the making of bronchograms must be carried out with a detailed plan and technique. The patient must be psychologically prepared ahead of time. Adequate anesthesia

must be obtained. This is not the same for every patient and may require the use of various anesthetic agents in different patients. In some patients the application of topical anesthesia within the tracheobronchial tree is adequate. In some patients, particularly bronchiectatics, it is necessary to empty the bronchi by bronchoscopy and suction and to apply more anesthetic agent to the areas laid bare by the removal of secretion. In some patients it is necessary to give drugs such as codeine by vein during the course of the bronchoscopy to decrease the desire to cough. In an occasional nervous patient the anesthesiologist may administer a sedative dose of a barbiturate intravenously during bronchoscopy. In an adult it is not desirable to use enough of this drug to induce sleep but one can use a smaller quantity to induce sedation with distinct advantages at times. The injection of the contrast medium should be done under fluoroscopic control and preferably with cooperation between the radiologist and the bronchologist. In some hospitals the radiologist is too busy to do this work or lacks sufficient interest in it. In such cases the bronchologist may be forced to do the entire procedure. We have been fortunate in having a great deal of interest on the part of our radiologists and as a result the quality of the bronchographic studies has continually risen until we now are completely dissatisfied with anything except a very adequate demonstration of all major and most minor bronchi. This effort has enabled us to demonstrate small areas of bronchiectasis as described by Overholt and Walker, forming a basis for recurrent asthma or pulmonary bleeding which we would have missed completely in years past, and which we have reason to believe are missed now in less exact studies of the bronchial tree. Recently several techniques for the making of bronchographic studies in children have been described.[6,7] These require careful planning but they permit entirely satisfactory bronchographic studies at almost any age (Figs. 1 through 4).

Not long ago Overholt[8] published a comment on the diagnosis of early pulmonary cancer and the relationship of the various branches of medicine concerned with this diagnosis. His statements are well worth examining and every person doing bronchology should consider the ideas he puts forth. His primary tenet is that the positions of some lesions in the lung make their histologic diagnosis impossible except through biopsies obtained on thoracic exploration. He believes that sometimes lesions which are clearly visible by x-ray are passed over by the family doctor, the internist or the bronchologist

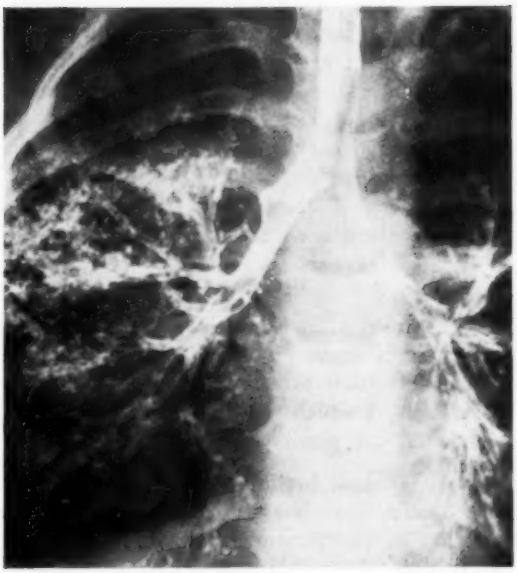

Fig. 4.—Bronchogram of a three year old boy showing a completely normal right lung except for total stenosis of the right upper lobe bronchus. All major branch bronchi can be fairly well visualized in this film. In the films of the series of bronchograms all these bronchi could be seen. The left major bronchi are also visible in this film although special views are necessary to separate one from the other.

as being unimportant when the bronchoscopic findings seem normal. He feels that repeated bronchoscopy is sometimes carried out in such cases in a fruitless effort to determine the type of lesion and that valuable time is lost to the patient before surgery is finally done. With this point of view the author is entirely in accord. Our procedure in Minneapolis has been to have all cases studied by the bronchoscopist and the thoracic surgeon. When an internist or family doctor refers a patient for bronchoscopy the thoracic surgeon is always consulted before or after the bronchoscopic studies have been carried out. We do not subscribe to Overholt's idea that in most instances bronchoscopy can be most advantageously carried out on the day of thoracic exploration. We feel that it is advisable

to study the case ahead of time and to get all information possible. It is an advantage to the thoracic surgeon if cell studies have been made and if a biopsy has been made and paraffin sections rather than frozen sections studied. On the other hand, we certainly agree that loss of time due to such studies should be minimal. We frequently see patients, bronchoscope them, find that we cannot make a diagnosis by the bronchologic study, and immediately recommend thoracic exploration. If the patient accepts this recommendation his exploration can be carried out within two or three days of the time he starts his study and there is no significant loss of time.

SUMMARY

Precisely performed bronchology will forearm the thoracic surgeon with information about his patient which cannot be obtained otherwise. This "precision bronchology" will include many small individual parts each of which must be well done to make the final product as good as it can possibly be.

The parts will include preliminary study of the patient by internist, family doctor, bronchologist and thoracic surgeon; psychological preparation of the patient; careful bronchoscopy including telescopic observation of bronchi, aspiration of specimens, bronchial measurement; the careful preparation of bronchograms by the bronchologist working with the radiologist and a group of x-ray technicians prepared to work quickly and efficiently; and finally a summing up of the problem with final disposition of the patient made after consultation between bronchologist, radiologist and thoracic surgeon; such disposition not causing undue delays which may prejudice the patient's chances for recovery because thoracic exploration is not promptly performed.

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XLIII

THE SURGICAL COMPLICATIONS OF DIVERTICULUM OF THE PHARYNX

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The complications of any operation often determine its success or failure. Surgeons, being influenced by the frequency and severity of complications, develop the best operation by learning to avoid complications. This applies to the development of the surgical treatment of diverticula of the pharynx, which is related to the progress of radiology, esophagology, anesthesiology, and the antibiotics. In spite of great progress, complications are still of considerable importance in present day surgery for pharyngeal diverticulum, even though little mention of this is made in most recent articles.

Diverticulum of the pharynx has interested many medical authors since the first reference to it by Ludlow of England in 1764. His original specimen is still in the Hunterial Museum of Glasgow. In 1840, Rokitansky studied this condition and considered diverticulum of the pharynx as due to internal pressure or pulsion in contradistinction to diverticulum of the esophagus proper, which is produced by external pull or traction. In 1877, Zenker found twenty-two cases reported in the literature and added five of his own. He correlated the patient's symptoms to the pathological findings and established diverticulum of the pharynx as an organic entity. Since this time the term Zenker's Diverticulum has been used by many to designate this condition. Negus[6] clarified the etiology of pharyngeal diverticulum by his study of comparative anatomy.

The first operation, excision of the sac, was performed by Kunge in 1864, the second by Hamburger in 1871. Both patients died operative deaths.[1] The third operation, performed by Niehaus in 1884, resulted in death from hemorrhage on the twenty-fourth postoperative day. Not until 1885, twenty years after the first attempt, was this operation successfully performed by Wheeler of Dublin, resulting in a cure.

During the next twenty-five years considerable progress was made. Stetton,[2] in 1910, collected from the literature all the cases in which operations had been performed, amounting to sixty. The death rate was "only 16.6%." In the group of forty-eight cases having direct excision of the sac, the mortality was 18.7%. The most common complications were infection, mediastinitis, pneumonia, and cervical cellulitis. Stetton believed a preliminary gastrostomy was advisable, as it solved the post-operative feeding problem. He was not impressed with the two-stage operation, invagination of the sac, or destruction of the mucosa of the sac. His paper taught the surgeons to fear the complication of infection in the one-stage operation, so other procedures were developed.

Two-stage operations were reported by Goldmann, Mayo, Murphy, Lahey, and others. Invagination of the sac was reported by Halstad, McClure, Girard, and others. Imperatori, in 1927, inverted and excised the sac by endoscopic means, and diverticulopexy was reported by Hill and Konig. Mosher reported endoscopic division of the common wall between the sac and the esophagus, but gave it up because of the danger of mediastinitis. Gaub and Jackson, in 1915, devised a combined esophagoscopic and surgical method, and in 1926, Jackson and Shallow[3] reported a series of fifteen operations performed by this method. One patient died of postoperative massive collapse of both lungs. These authors state that the two-stage operation has been complicated by insufflation pneumonia, mediastinitis, secondary hemorrhage, fistula, and recurrence of the sac. They successfully avoided the complication of infection in a one-stage operation.

The esophagologist can aid the surgeon by locating the sac and deciding on which side of the neck the incision should be made, and by emptying the sac before it is excised. He can place the end of the esophagoscope into the sac, thus positively identifying the structure. This maneuver has been of particular value to the surgeon when the tip of the sac extends below the clavicle.

After the sac is pulled up, the esophagoscope can be inserted into the esophagus below, thus helping the surgeon to amputate the sac in the place least likely to be followed by postoperative stenosis.

Jackson and Coates'[5] book "Diseases of the Nose, Throat, and Ear," published in 1929, contains an article by Mosher stating that the writer considered the one-stage operation unjustifiable as it had

a mortality of 20%. Jackson and Jackson's, "Diseases of the Nose, Throat, and Ear," published in 1945, states that there should be no mediastinitis, no persistant fistula or sinus, and no stricture with the one-stage operation. With the two-stage operation laryngeal paralysis occurs in 9%, permanent fistula in 9%, incomplete removal of the sac in 36%, and stenosis in 9% of the cases. Shallow and Clerf,[7] in 1948, reported 186 cases operated upon by the combined one-stage method. Mediastinitis was prevented. In the last 100 cases, nine patients developed unilateral vocal cord paralysis, seven had minor wound infections, four had parotitis, one fistula, one atelectasis, one pneumonia, and two heart complications.

In 1928, Kennedy[1] and I reported the first successful operation done by the combined method in our community. Following this, I have seen 23 additional cases, which make up this report. All are private cases. The surgery was done by 13 different surgeons. General anesthesia was used in all. Infection did not occur in any case, so this once frequent and often fatal complication has now been successfully eliminated. Better surgical technique, including pre- and post-operative care, better anesthesia, more effective antibacterial agents, and earlier diagnosis are all responsible for this improvement. However, other complications did occur.

Fistula, of a temporary nature, occurred in three patients. All closed spontaneously. This is worthy of mention, as the surgical method of closing the stump, varied from careful suturing, layer by layer, to no suturing of any kind, but merely applying a clamp and allowing it to slough off in a few days, according to the method of Maxeiner.[4]

Stricture at the site of removal of the diverticulum occurred once. A few dilations relieved this patient promptly.

Recurrence of the sac was noted twice. It seems that this might well happen, as the original causative factors have not been altered by the operation.

Chest complications occurred in three patients; (1) unilateral pneumothorax, (2) post-operative atelectases and (3) post-operative aspiration of vomitus, requiring immediate suction of the bronchi. All recovered.

Unilateral vocal cord paralysis occurred in two cases. One was that of a large sac extending into the chest and had a pack of oxy-

cell left in place to control hemorrhage. The other occurred in a two-stage operation. Both recovered spontaneously.

Failure to find the sac, in one case, by both the surgeon and the esophagologist. The sac was still present post-operatively in x-ray.

Pre-existing complications in four cases contraindicated operation:

(1) Bulbar paralysis, as well as diverticulum. A gastrostomy was done and successful feeding was carried out for several months.

(2) A fish bone lodged in the sac which required removal, but no surgery for the diverticulum was desired by the patient.

(3) A serious cardiac condition.

(4) A nasal suction tube was needed after a major surgical Procedure. About forty-five minutes was spent searching unsuccessfully with the esophagoscope for the subdiverticular orifice. Any time a patient with a diverticulum has major surgery, he should swallow a thread previous to the operation. The thread would serve as a guide in passing the suction tube should the need occur.

Excision of the diverticulum should be advised even though it is small and the symptoms seem insignificant. The sac will surely enlarge, increasing the patient's discomfort until operation is a necessity. As a general rule, the smaller the sac, the easier the operation is on both the patient and the surgeon.

The earlier patients were all fed postoperatively through a transnasal tube left in place in the esophagus. After observing some patients who pulled the tube out, it was found that they did as well as those who kept the tube in place. The use of the feeding tube is now eliminated and the patient is fed intravenously for the first few days.

Infection has been practically eliminated by the antibiotics. Fistula, stricture, vocal cord paralysis, and recurrence should be preventable by earlier diagnosis and operation while the sac is small; by better preoperative care; by better surgical technique, including gentle manipulations of the strictures; by putting less pull on the laryngeal nerves; and by accurately suturing the mucosa and muscle layers. Chest complications will be minimized when the anesthesia is carefully given and extra attention is paid to the air way to keep it completely clear. Elimination of the feeding tube is recommended.

Undoubtedly, the development of better teamwork for this operation would help avoid complications. The latest articles on the surgery of pharyngeal diverticulum make no mention of the combined operation. Some surgeons may find that they do better without any internal manipulations, though none has so stated. None has stated that he feels the use of the esophagoscope is a cause of complications.

CONCLUSIONS

Twenty-four cases of diverticulum of the pharynx are presented of which twenty were operated upon.

No death occurred, but twelve patients had some serious complication develop and eight had smooth, uncomplicated recoveries.

No infection such as mediastinitis or cervical cellulitis occurred.

Surgery should be advised as soon as the diagnosis is made.

Postoperative feeding tubes can be eliminated.

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XLIV

REALISM IN THE SURGERY OF THE TONSILS AND ADENOIDS

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The concept governing tonsil and adenoid surgery has undergone important and radical changes in recent years. Historically, the management of the tonsil and its allied problems has always reflected the evolutionary developments characterizing the various epochs in medicine. The first record of tonsil surgery occurred as far back as the year 1000 B.C. The operative procedure was extremely primitive and consisted of the removal of one-third of the tonsil with a curved knife, the tonsil being held by means of forceps. Fear of fatal hemorrhage prevented a more extensive operation. Thus, even in this early era of the development of medicine, it is apparent that the tonsil presented a serious medical problem recognized as such by the pioneers and treated surgically by the best available weapons at their disposal. A few centuries later, we find that the need for the complete removal of the tonsil was recognized, the procedure being to tear the tonsils out with the fingers. Only in the presence of marked adhesions, was cutting resorted to. The first instrument designed for the enucleation of the tonsil was the ordinary spoon with a hole in the center. According to historical legend, it was devised by a Norwegian peasant.

The guillotine is a comparatively recent innovation. It has had a tremendous impact on tonsil surgery. It has facilitated the successful removal of the tonsil by well-trained operators. In the hands of untrained operators, it has yielded poor results. The simplicity of its use has encouraged many untrained individuals to employ it on a large scale. At the present time, at least fifty per cent of the cases are operated upon by individuals unqualified by training to perform this operation. Furthermore, it has focused the spot-light on the operative procedure at the expense of the broader concept underlying the pathogenesis of infections of the pharyngeal lymphoid tissue.

The pharyngeal lymphoid tissue plays an important role in acute infections of this area. In chronic infections, it assumes even greater importance as a possible source of focal infection. Then it must always be recognized as a factor leading to middle ear diseases, either by direct extension or secondarily by obstruction of the eustachian tube with subsequent ascending infection. It is obvious that the successful surgical treatment of infected pharyngeal lymphoid tissue requires the complete extirpation of all such infected tissue. Partial removal limited to the tonsils or even the tonsils and adenoids fails to recognize the anatomical and pathological entity of the lymphoid structures in this area. Complete extirpation should include the faucial tonsil, the lingual tonsil, the pharyngeal tonsil and all secondary lymph nodes.

The faucial or palatine tonsil offers the surgeon the least difficulty, since it can readily be visualized and offers a ready approach for its removal. Unfortunately, the importance of the preservation of the posterior pillars during operative manipulations is not fully recognized or emphasized. Eliminating them destroys the symmetry of the pharynx, causing the palate to be distorted and effecting the modulation of the voice. Destruction of the anterior pillars usually or often results in extensive scar tissue accompanied by a feeling of tension and discomfort.

The lingual tonsil has come to the front only during recent years, as a surgical problem. Many factors explain this delayed evaluation of its importance in infections and focal infections. It is not readily accessible for routine examination so that it ordinarily is not considered in its true role. It also presents many problems for its operative removal. Treatment by radiation was usually resorted to in the past for obvious reasons. At the present, with the development of suitable instruments, the successful removal of the infected lingual tonsil can be accomplished in all cases, and should be done routinely at the time of the removal of the faucial tonsil, when found to be necessary or desired.

In our experience, at least 85% of tonsillectomized patients require the removal of concomitantly infected lingual tonsil nodules. Anatomically, the lingual tonsil presents the same picture of infected cypts and pockets as characterizes the palatine and the pharyngeal tonsils. During operations, exudative products of inflammation can be readily extruded from such pockets by the application of pressure.

On gross examination, the experienced surgeon can readily discern the presence of inflammation. The lingual tonsil tissue extends across the base of the tongue and inferiorly to the floor of the valecula. Upon its complete removal, a comparatively smooth, fresh surface reaching down to the epiglottis is formed. With tonsil scissors, that portion of the lingual tonsil adjacent to the lower pole of the faucial tonsil is dissected free from the underlying tongue muscle. The lingual tonsillotome and the snare are then employed to extirpate the lymphoid aggregates lying posteriorly and medially. When removed, the lymphoid nodules, varying in size up to several millimeters in diameter, are held together in a loosely arranged clump by the adjacent mucous membrane and its matted fibrous stroma. The use of a suitable tongue depressor determines the ease and success of the operation. Many of the instruments marketed for this purpose are entirely unsatisfactory. Extreme flexibility is a prime requisite. Provided the underlying muscular layer of the tongue is avoided, there is very little blood loss.

In the case of the pharyngeal tonsil, the surgical problems are even more extensive. Until recent years, the end results were poor. This could be attributed to the use of inadequate instruments, the lack of proper visualization of the surgical field and of a good approach. The value of removal of adenoids could, therefore, not be estimated with any degree of certainty or accuracy. As previously stated, this aggregation of lymphoid tissue plays the same role in acute and focal infections as the faucial and lingual tonsils. Crypts and pus pockets may be present throughout the entire structure. Furthermore, due to its location, it plays an additional role in middle ear infections. The concept governing the removal of the pharyngeal tonsil as outlined above is an evolution of an experience dating back for the past 15 years. It was soon recognized that the lymphoid tissue occupying this anatomical area was much more extensive than is generally understood. The need for instruments for better visualization of the operative field, with particular reference to the pharyngeal recesses, soon became apparent. The type of instrument employed for the removal of the diseased tissue also underwent a gradual development. At first, following the usual operation with the La-Force adenotome, we employed the punch forceps to remove the adenoid tissue in the lateral pharyngeal recesses. The Yankauer nasopharyngeal speculum afforded a very satisfactory approach. A series of punch forceps were evaluated for this purpose. The first punch

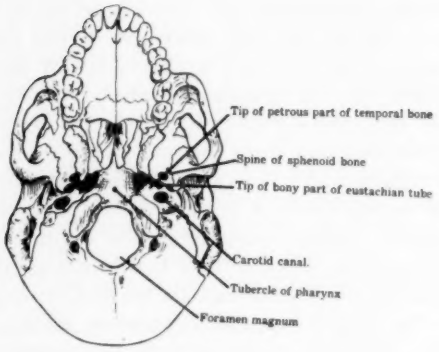
Tip of petrous part of temporal bone
Spine of sphenoid bone
Tip of bony part of eustachian tube
Carotid canal
Tubercle of pharynx
Foramen magnum

Fig. 1.

forceps was the Hartman round ring forceps on the universal handle. It was succeeded by a triangular type of forceps which in turn was superseded by the use of three types of basket forceps, a large and a medium sized triangular forceps and a small elliptical forceps. With the basket type forceps, by the use of two or three sizes, it was soon discovered that the tissue could be more readily and accurately removed. We found that by placing the tip of the Yankauer speculum behind the mouth of the eustachian tube, it could then be more readily displaced forward, thus affording a much wider view and more adequate approach to the pharyngeal recesses. The following technique was finally adopted and is now used for the removal of the adenoid tissue. The lymphoid tissue is grasped with the basket forceps of choice and, by rapid biting, not attempting to take too large a bite, in any case, more frequently not making a complete bite but instead taking hold of small bits of tissue and stripping them off the base, the tissue is separated and removed with frequent application of the suction tube. With alternate use of the forceps, a suction tube and stick sponges, all the tissue is removed down to a clean base which can be verified by direct inspection after the hemorrhage is controlled. By this method, all the lymphoid tissue extending to the lateral pharyngeal walls can be removed. The lymphoid tissue on the lateral walls will be found to cover the base diffusely and does not occur in nodular aggregates as one would ordinarily expect. Bleeding is readily controlled by pressure with sponge sticks.

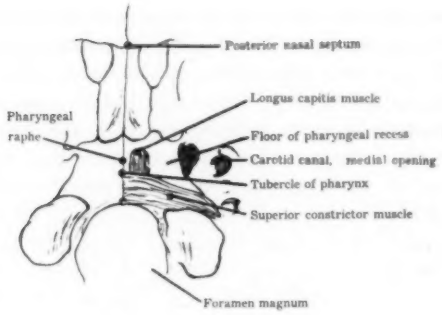

Fig. 2.

The wide limit to the lateral extension of the pharyngeal recesses, which we have found to exist while operating on these cases, is substantiated by reference to texts on the anatomy of this area. Cunningham's text on anatomy establishes the extent of these spaces by designating three points of attachment which are demonstrated in Figure 1. They are the tip of the petrous part of the temporal bone, the spine of the sphenoid bone and the tip of the bony part of the eustachian tube. The anatomists tell us that, because of changes that occur in cadaveric material, a correct impression of the size of the pharyngeal recesses cannot be obtained from such material. We have found, at operation, that by extending the removal of tissue to the anatomical points given above, a relatively large space is encountered. We have confined the removal of tissue to the posterior wall of the space and avoided traumatizing the mucosa around the opening of the eustachian tube as much as possible, for the obvious purpose of keeping down adhesions. This situation is more readily appreciated when we consider that the opening of the eustachian tube is related to the anterior wall of the pharyngeal recess, being in the neighborhood of an inch nearer the midline of the pharynx than the lateral points of attachment of that structure as we have shown on the anatomical illustration. The so-called tubal tonsil is not in the mouth of the eustachian tube, as it is generally believed to be, but it is, according to Cunningham's Anatomy, a lateral prolongation from the pharyngeal tonsil onto the posterior wall of the pharyngeal recess. Our observation is in accord with the view as just stated.

The posterior lip of the eustachian tube is frequently hypertrophied, but we have not observed it to be the base for the growth of lymphoid tissue as generally believed. The lymphoid tissue, which is frequently found covering the salpingopharyngeal fold, is objectionable and should be removed if very large but because of its remoteness from the eustachian tube which points in the opposite direction, would obviously have very little influence on that structure. At the conclusion of the operation, the anatomical markings that can be observed are shown in Figure 2. The upper lateral part of the floor of the pharyngeal recess extending inferiorly as far as the eustachian tube is the aponeurosis covering the occipital bone. The lower part of the area is occupied by the superior constrictor muscle. Lateral to the pharyngeal raphe and above the superior constrictor is the longus capitus muscle. The raphe, in the midline, as stated above, shows a considerable depression at the lower part, marking the area between the anterior arch of the atlas and the base of the occipital bone. This depression marks the position of an increased depth of the adenoid body and an increased likelihood for formation of focal infection points and sometimes frank abscesses. The lateral walls of the pharyngeal recesses are loosely attached to the under surface of the petrous part of the temporal bone. The fibrous wall on either side bridges over the space between the lateral margin of the base of the occipital bone and the points of attachments of the lateral pharyngeal wall, as previously stated, viz., the tip of the petrous part of the temporal bone, the spine of the sphenoid bone and the tip of the bony part of the eustachian tube. The pulsations of the carotid artery can frequently be observed following the removal of the lymphoid tissue in this space.

The technical difficulties in this operation are greatly enhanced by the tendency to profuse bleeding which, however, can be soon controlled, since it is usually due to capillary oozing and not to vessels of any large proportions. The Yankauer speculum has given us the best approach, due to its flexibility and the small diameter of its lumen. By traversing the oral cavity at an angle of 45 degrees, with this instrument, the posterior lip of the eustachian tube can be easily retracted foreward away from the posterior pharyngeal wall with the lip of the instrument, as previously stated and the entire field made accessible to operation under direct vision. Instruments for retracting the palate, having larger diameters than the Yankauer speculum, are obviously less effective in reaching the higher areas.

My experience lecturing to senior medical students at the University of Tennessee impresses me with the fact that this is the year that is most favorable to present to them the concept of good tonsil and adenoid surgery. They can readily understand that it is a painstaking and time consuming procedure and because of its magnitude will require a relatively long period of training for those who are to enter this field of surgery.

Realism in tonsil and adenoid surgery confronts the otolaryngologists inescapably, with the responsibility of maintaining a high standard of training and practice in this field. The evaluation of this procedure has in the past depended upon its evolutionary changes in attempting to deal adequately with the diseased lymphoid tissue of the pharynx. The same evaluation must continue to be the criterion of the operation, if it is to be accorded its relative value in the treatment of diseases of the pharynx.

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XLV

STUDIES OF THE OTIC LABYRINTH

IV. ON THE DEVIATION OF THE VISUAL AXES (AS EVIDENCED BY THE MADDOX ROD TEST) AFTER THE BARANY TURNING TEST

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It has been found that the visual axes deviate in some individuals following vestibular stimulation by means of the Bárány turning test.[3] The question arises as to whether this deviation results from the stimulation of the vestibular organs or whether it is due to something else.

The oculogravic illusion recently discussed by Graybiel[4] is, in part at least, a labyrinthine phenomenon, altho Graybiel does not emphasize that feature. This phenomenon occurs when a subject is seated upright on a revolving turntable some five meters from the center. It results from a combination of stimuli: visual, pressure upon the skin from centripetal force and the force of gravity, from the action of the same forces upon the otoliths of the utricle and saccule and upon other organs and tissues in the body. Graybiel feels that the semicircular canals do not enter in unless acceleration of rotation is too fast. The character of the illusion depends upon which sense dominates. It is usually characterized by a displacement in the environment and a sense of tilting of the body. Rotation of the environment may be elicited under certain circumstances. Rotation and displacement may occur simultaneously.

When a subject is being rotated in a turning chair, his head tends to turn in the direction opposite to that of rotation. This is a manifestation of the righting reflex, which reflex is a labyrinthine phenomenon.

The complicated compensatory fixation reflex,[8] by means of which the eyes can be maintained in continuous fixation upon a stationary object in the environment during movements of the head, is another labyrinthine function. It is a postural reflex concerned in maintaining equilibrium and dependent upon the utricle and saccule, the semicircular canals and the muscles of the neck and back.

Work done as visiting research professor at Columbia University, Department of Otolaryngology, and supported in part by grants from the Research Council of the American Otological Society and by the Hayden-Coakley Memorial Fund.

Post-rotational nystagmus, during which the environment rotates, is labyrinthine, and sometimes has practical effects.[5] There are other labyrinthine effects, such, for instance, as the effect of labyrinthine stimulation on the fusion frequency of flicker[11] and the effect upon retinal after-images.[6, 7]

The righting reflex, the compensatory fixation reflex and nystagmus are all involved in the Bárány turning test and are all under labyrinthine control. It is only natural to assume that the displacement of the image due to deviation of the visual axes resulting from stimulation of the same ocular muscles which are producing rotation of environment, at the same time and under the same circumstances, is also due to labyrinthine activity. More especially, perhaps, since the effect lasts an appreciable length of time after the cessation of turn. However, under the fusion free conditions of the Maddox rod test, it is not necessarily true that this deviation is also due to labyrinthine stimulation. Fixation, accommodation, convergence and attempts at fusion[1] also enter in with other reflexes and certain voluntary movements, which are not under labyrinthine control.* Any or all of these reflexes might combine to cause the deviation measured by the Maddox rod test.

The Maddox rod test is used in ophthalmology for demonstrating the balance or imbalance of the extraocular muscles as evidenced by the mutual relation of the visual axes in the two eyes.[10] It is carried out as follows: The subject is asked to fix (look at) a small, bright white light. Then a small glass rod, suitably mounted, usually red in color, is placed before one eye. The white light, when viewed through this rod, appears as a red streak. When the rod is placed horizontally before the eye, the streak appears vertical and when the rod is vertical the streak appears horizontal. The Maddox rod serves to break visual fusion because the image received from the eye covered by the rod is so disguised that it is not identified in the visual center as the same light fixed by the fellow eye. When fusion is thus broken, the extraocular muscles no longer hold the visual axes parallel. The eyes tend to move toward the position of rest. More accurately speaking, they move toward the "fusion free" position.**[1] If

*Perlman and Case[9] give an excellent discussion of the inter-relationship between voluntary eye movements controlled by the frontal cortex, reflex optic movements which are controlled by the occipital cortex, activated by retinal reflexes, and the reflex vestibular movements, which are controlled in the cerebellum and activated by the end-organs in the labyrinth. These inter-relationships must be understood before eye movements can be analyzed.

**The "fusion free" position does not usually coincide with the position of rest. The position of rest is attained only in sleep or possibly even only after death.

the eyes are orthophoric—in perfect balance—the axes will remain parallel and the red streak will be seen to bisect the white light. If the eyes are heterophoric—i.e. if there is a latent imbalance—then the streak will be seen in some other position. If the eyes diverge, the streak will cross to the other side; i.e. if the rod is before the right eye, the streak will appear to the left of the light. If the eyes converge, the streak will be on the same side as the rod—in this case, to the right. The deviation can be measured by placing appropriate prisms before one or both eyes until the streak is again seen to bisect the light. The amount of prism required is a measure of the deviation.

The Maddox rod test is widely used because it is so simple, but, unfortunately, it is not dependable as a measure of phorias. It measures the deviation with great accuracy but does not label the cause of the deviation and there are other causes of deviation. Therefore, the careful ophthalmologist, seeking to measure some latent muscle imbalance, checks his Maddox rod findings with other tests.

Convergence and accommodation are both under voluntary control. The two are so closely related in action that they cannot be separated with facility. They are habitually used together. Accommodation is habitually accompanied by appropriate convergence in order to keep both eyes directed upon the object which is being observed (fixed). The other vergences—divergence, left and right hypervergence—are not under voluntary control. When fusion is broken by the Maddox rod and the subject sees two images—a white light and a red streak—he tends to be confused. It is difficult to keep both in view at the same time. The eye covered by the Maddox rod tends to suppress and then the red streak to disappear. The locus of the red streak in space is indefinite and in attempting to locate it and "fix" it, the subject often accommodates (focuses) and this results in an associated convergence. As a result of the convergence, the red streak, in turn, shifts position. As the subject changes his accommodation and degree of convergence in trying to locate the streak, the position of the streak in relation to the light also changes. This shifting is apt to be accentuated if the subject attempts to fix the streak rather than the light. He may also attempt to fuse. Changes in fusion cause rather slow movements in the visual axes.[7] All of these factors combine to cause the streak to wander back and forth in an uncertain but sedate manner, disappearing entirely from time to time. Many patients will tell the examiner, "It (the streak) won't stand still." I have even had one astute patient say, "This test can't mean anything, I can put that streak anywhere I want to!"

If, under these circumstances, further confusion is added by the nystagmus and vertigo following turning, the resulting movements of the red streak (deviation of visual axes) must be very carefully evaluated before any significance, as far as the labyrinth is concerned, is attached to them.

The present study is made for the purpose of determining if the deviation which occurs following turning in the Bárány chair is due to labyrinthine stimulation. Another feature studied is the possible effect upon the vertigo of wearing prismatic lenses before the eyes. It has been stated that small prismatic lenses will control the subjective sensation of vertigo.[3, 12]

OBSERVATIONS

The subjects used in these tests were with two exceptions freshmen medical students, and these two were patients.

The Maddox rod test was used in the usual manner with this variation: The white light had been placed in the center or zero position of an arc constructed of plastic board (masonite) upon which the deviations in prism diopters had been previously marked and which was so placed before the turning chair that the subject faced it when the chair was at rest. As the subject reported the position of the streak on the arc, the deviation could be read off directly in prism diopters.*

The test was made under the conditions of four different situations:

(A) Control conditions before turning in the Bárány chair; (B) immediately upon stopping the chair at the beginning of the period of post-rotational nystagmus and vertigo; (C) at the end of the period of post-rotational nystagmus and vertigo and (D) some of these repeated with the subject wearing a small prism before his eyes, placed with the base in.

During the control tests (A), the subject was instructed to fix the white light and the red streak alternately. He was urged especially to get the red streak into clear view. This either caused attempts at fusion or caused his accommodation and convergence to vary somewhat. In any case, the red streak varied in position. The resulting variations in the angle between the visual axes were determined by reading off the position of the red streak on the arc as reported by the subject.

*It is to be noted that this is a measure of total deviation only and does not necessarily correspond with any phoria present.

During the post-rotational tests, the subjects were instructed to look at the white light always and these readings were compared with those made in the control tests when the white light was fixed. In the tests made immediately upon stopping the chair (B), the subject was instructed to look at the white light as soon as the chair had stopped and report the position of the red streak. All found this difficult to do because of the nystagmus but most managed to report satisfactorily. Meanwhile, the operator started the stopwatch immediately upon cessation of rotation, for the purpose of timing the nystagmus. The subject reported the cessation of vertigo and this was timed also.

When both nystagmus and vertigo had ceased, another reading of the position of the red streak was made (condition C).

(A) Nine control tests were made on seven different subjects. With the Maddox rod in place before the right eye, the subjects were told to fix alternately the white light and then the streak. Convergence resulted in looking from the light to the streak in six of the tests. The amount varied from 1 prism diopter to 7 prism diopters. Four of these were 1.75 prism diopters or more. Divergence occurred in three. One diverged 1 prism diopter and the others 2 prism diopters each. The convergence averaged 2.85 prism diopters and the divergence 1.75 prism diopters.

(B) There were 25 determinations of change in visual axes taken immediately upon cessation of rotation at the beginning of post-rotational nystagmus and vertigo with the subject fixing the white light. In 13 a convergence was found, in 12 there was no deviation. Of the 13 in which convergence was found, the change consisted of increased esophoria in 7 and a decreased exophoria in 3. In 2 a small exophoria became an esophoria. In one there was no record of the phoria although the amount of change and the direction were given. The order of magnitude of change, 1 to 8 prism diopters, was about the same as in the control tests—1 to 7 prism diopters. The average was 3.50 prism diopters.

(C) There were 24 turning tests made in which the Maddox reading was made both at the beginning and at the end of the period of post-rotational nystagmus; i.e. immediately upon stopping the chair and after the nystagmus had ceased. There was a divergence, during this period, in 12 and no change in 10. In only 2 was there a convergence. One (.25 prism diopter) was within experimental error and probably the second also (1 prism diopter). All exhibited nystagmus for periods varying from 22 to 30 seconds—those which indicated no change as well as those which diverged.

(D) It has been claimed[12] that a small prism base in controls or decreases vertigo under certain conditions.

Aschan[2] tested 100 candidates for military aviation, using the Bárány test with and without Frentzel lenses. He found that the post-rotational nystagmus was substantially increased by wearing the glasses. He does not state specifically if there was any effect upon the subjective vertigo. Frentzel glasses are strong convex lenses, which blur the vision to the point where fixation is abolished. Such convex lenses would give a prismatic effect if the distance between the optical centers of the lenses were different than the interpupillary distance of the subject wearing the lenses. Aschan does not discuss the possibility of such a prismatic effect on the subjective vertigo nor does he suggest it as a factor in the variation of the duration of the nystagmus.

Small prisms (1.5 to 2 prism diopters) were worn before the eyes in 12 of these turning tests. In 11 the base was in and in one the base was out. In these 12, the subjects reported that the vertigo after turning was less in 3, greater in 5 and unchanged in 4, as compared with previous tests made without prisms. The nystagmus was a trifle longer in 6, a trifle shorter in 4 and the same in 2. These results were considered to be without significance. These experiments gave no indication that the prisms had any effect upon the vertigo or the nystagmus. The prisms, of course, under all circumstances, changed the deviation reading by the amounts of their respective powers.

DISCUSSION

A deviation of the visual axes, resulting in displacement in the environment under the conditions of the Maddox rod test, occurs in some individuals after rotation in a Bárány turning chair. Nystagmus also occurs resulting in rotation of the environment. Thus, as in the oculogravic illusion, both rotation and displacement of environment may occur simultaneously. However, if the amount of stimulus is known, the oculogravic displacement can be predicted approximately as can the duration of the nystagmus. The displacement due to deviation of the visual axes, as in these turning tests, is not predictable. It occurred in only about half the subjects.

If this phenomenon were a result of labyrinthine stimulation, one would expect it to occur in normals each time the labyrinth is adequately stimulated. The stimulation in the Bárány turning test as used in these experiments is not only adequate but is in fact rather violent. If this deviation were due to labyrinthine stimulation, one would expect it to parallel the other signs of stimulation such as nystagmus and vertigo, at least roughly. This it did not do since it

failed to appear at all in about half of the tests. Vertigo and nystagmus occurred in all.

Moreover, in control tests made before rotation, the displacement of the red streak to about the same extent as that which followed rotation was produced merely by asking the subject to "look hard" at the streak and then at the light. This served to encourage accommodation. The shift during turning, as indicated by readings taken immediately upon stopping the chair, if it occurred at all, was always in the direction of convergence (the only voluntary vergence), as though the subject were accommodating or attempting to fuse. The shift which occurred during the post-rotational period, as the nystagmus and vertigo subsided, was in the direction of divergence—in other words, relaxing convergence. There were two exceptions to this both of which were in the range of experimental error.

The question naturally arises, why did not convergence occur in all? One can only say that probably all did not accommodate (or attempt to fuse). These are voluntary functions. There was no need to vary the accommodation since the light always remained at the same distance. Yet, in attempting to see, under difficult or unusual circumstances, many subjects do vary the accommodation even when the distance remains constant.

CONCLUSION

The deviation of visual axes following the Bárány turning test as evidenced by the Maddox rod test does not appear to be a result of stimulation of the labyrinth. Rather it seems to be a manifestation of the accommodation-convergence or fusion reflexes, or both of them.

626 Medical Arts Bldg.

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XLVI

STUDIES ON THE OTIC LABYRINTH

V. THE POSSIBLE RELATION OF THE INSERTION OF THE TECTORIAL MEMBRANE TO ACOUSTIC TRAUMA, NERVE DEAFNESS AND TINNITUS

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The mechanism of acoustic trauma or nerve deafness and the tinnitus which results has not been adequately explained. The steps of the pathology involved in these two conditions are not well understood, although there has been much study of both—experimentally, clinically and in postmortem pathologic sections.

Lurie[12] and his co-workers have produced definite changes in the cochlea with intense sound. These varied from slight degenerative changes in the hair cells to such severe damage that the cells on both sides of the basilar membrane were thrown off.

Wever and Smith[20, 21, 23] studied the histologic changes in stimulation deafness produced by various tones (300, 1000, 5000 and 10,000) and studied the distribution of the trauma. Schuknecht[18] has recently published an article on the effects of direct mechanical trauma.

Nerve deafness has been studied for a long time by many workers, especially that due to noisy occupations and gunfire. (Habermann,[9] Sacher,[16] Uffenorde,[22] Larsen,[11] Fowler,[7] Crowe, Guild and Polvogt,[5] Campbell,[3] McCoy,[13] Cope and Johnson,[4] Boemer,[2] Shambaugh and Juers,[19] McMahon,[14] Atkinson,[1] Guild[8] and Fowler and Fowler, Jr.[6])

Tinnitus is said to be due to spontaneous discharges in the hair cells or nerve fibers—often a result of degeneration.

In my work at Columbia last year I dissected 121 temporal bones, studying especially the cochlea, and learned that the tectorial mem-

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Work done in part as visiting research professor at Columbia University, Department of Otolaryngology, and supported partially by grants from the Research Council of the American Otological Society and by the Hayden-Coakley Memorial Fund.

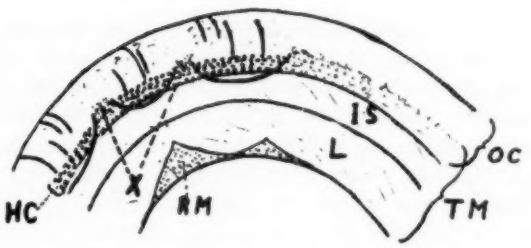

Fig. 1.—Diagrammatic representation of the tectorial membrane and organ of Corti in specimen 110. The outer margin of the tectorial membrane remains attached at the points X and is separated between these points and on both sides. The retraction of the separated portions is exaggerated for purposes of illustration. HC—hair cells. RM—remnant of Reissner's membrane. L—limbus. IS—internal sulcus. OC—organ of Corti. TM—extent of tectorial membrane.

brane has at least four different attachments: (1) the origin on the limbus, (2) an insertion upon the border cells of the internal sulcus along Hensen's stripe, (3) an insertion upon Hensen's cells along the outer margin and (4) an attachment to or contact with the hair cells.[10] In rare specimens, all of the attachments were intact when the cochlear canal had been exposed and opened (as in specimen 105 from a 68 old white female). The organ of Corti in this specimen could be lifted—in fact, the whole basilar membrane with all the structures atttached to it could be lifted and rocked back and forth by means of the tectorial membrane through its insertions. If these movements with the micromanipulator were anything but gentle, the insertion at the outer margin would tear (specimen 105 and specimen 102 from a white male 6½ months old). This insertion was found attached in a number of others but appeared to be in the process of tearing free. There were defects in it and the portions which remained attached looked as though they were under tension and about to let go (specimen 110, Fig. 1, from a six and a half months premature and 114 from a nine day old white infant). The outer margin lay entirely free in most specimens. Some of these showed shaggy and frayed remnants of the membrane or net of insertion both upon the cells of Hensen and upon the margin of the tectorial membrane (specimen 95, 64 year old white male). In others, with the outer border free, the organ of Corti could still be lifted gently by means of the tectorial membrane either through the

attachments to the hair cells or the inner insertion at Hensen's stripe (specimen 117 premature white female and specimen 108, colored male age 30).

The tectorial membrane was found floating entirely free from all attachments in some specimens in which the circular saw used for removal had torn the drum or passed through the middle ear dislocating the ossicles. For instance, in specimen 124 (73 year old white male) the saw entered the middle ear and just cut off the long process of the incus without doing any other apparent damage. Yet, when the cochlear tip was opened, the entire final turn of the tectorial membrane was found floating free of all attachments, including the origin upon the limbus. In an occasional specimen it was found loose with a loop hanging out through a tear in Reissner's membrane into the scala vestibuli, a condition which might be called evisceration of the cochlear canal.

Apparently it is quite possible to vibrate the ossicles (with the saw) so violently during removal of the specimen, that the tectorial membrane may be torn entirely free from all attachments. On the contrary though it is most difficult to remove the bone and dissect it so gently that all the attachments, including the insertion of the outer margin, are preserved, it is possible and was accomplished in some specimens.

From these dissections, it seems obvious that the insertions of the tectorial membrane do not tolerate very much stretching and that when they let go, they do so in a definite sequence. The outer margin first, then the hair cells and Hensen's stripe; after the insertions have all let go, then the origin also may become detached.

During life, the tectorial membrane is supported in place over the hair cells by the cells of Hensen and the border cells in such a way that there is no tension on the hairs of the hair cells in the absence of sound vibration; at least there normally is no sound (or tinnitus) in the absence of mechanical sound vibrations. If my concept[10] of the anatomy and physiologic action of this arch is correct, then only during sound vibration would the abutments extend enough to allow tension directly upon the hairs of the hair cells.

It is known from the experiments cited above and many others, that acoustic trauma, if sufficiently great, injures the organ of Corti severely and extensively. In the light of my dissections, it seems likely that trauma would first act upon the insertion of the outer border of the tectorial membrane and cause stretching or tearing. This is the most vulnerable connection. Such damage would not

have been apparent in any histologic studies because the outer margin of the tectorial membrane is invariably loosened during histologic preparation. If the insertion of the outer border were to be stretched or torn in such manner that the support of the tectorial membrane were transferred from this insertion to the hair cells, might it not be conceivable that there would be tension enough upon the hairs to cause a continuous stimulation which might result in tinnitus?

The explanation often given for tinnitus in presbycusis is that it is a reaction of degenertion. One wonders how the degeneration could be continuous over a period of 20 or 30 years, making stimulation all that time. Other possible explanations occur to one's mind. If, for instance, a slight mechanical stimulation, such as traction from abnormal tension just described, were present, might it not be possible that the stimulation could, under these circumstances, be continuous over several decades? Tinnitus actually does last over several decades. Some observed variations in tinnitus might support this view. For instance, the tinnitus sufferer may notice at times that there is a cyclic intensification which takes place during walking, synchronous with his footsteps. Pressure on the jaw or top of the head may also intensify it.*

Be that as it may, in dissection of the cochlea, the insertions of the tectorial membrane are usually torn, and that at the outer margin goes first. If one can assume that the sequence of events in the breaking of the insertions of the tectorial membrane from acoustic trauma is the same as was found in trauma from the dissections

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*Mechanically speaking, the otic labyrinth is a small, closed fluid-container with two yielding spots in the wall (windows covered with membrane). One of these soft, yielding areas is placed at a level 3 mm above the other. As a person walks, this little container rises and falls with each step. From the standpoint of physics, because of the inertia of the fluid, there would be movement between the two windows, where the fluid column stands 3 mm high. I have imitated this movement in a crude way in some of the specimens dissected and have found that the perilymph simply moves back and forth through the helicotrema and axially in the scala vestibuli. Perlman[15] has described similar axial movement from vibrations of a very low frequency. During these slow movements, one cannot detect any up and down movement of the basilar membrane and the structures upon it, although, from the standpoint of physics again, there must be some because no structure is absolutely rigid. Under normal circumstances, these minimal movements must be absorbed by the insertions of the tectorial membrane at the outer border and at Hensen's stripe, in which case they do not stimulate the hairs of the hair cells. But, if the insertion of the outer margin were defective and the support of the tectorial membrane fell upon the hairs or hair cells, then such minimal up ond down movements in the basilar membrane as would be caused by the swinging of the perilymph during walking, could stimulate the hair cells.

I have had a high-pitched tinnitus for more than 15 years and, during this time, have often noticed that there is a cyclic increase and decrease synchronous with the steps in walking. I have attempted to place the point of greatest intensity in the walking cycle—a tantalizingly difficult thing to do. As nearly as

(many of the specimens which were dissected in Study I were dissected fresh immediately after death) then the first damage which occurs from acoustic trauma would be to the insertion of the outer border of the tectorial membrane (we are considering now only damage to the internal ear). Increasing the intensity of the traumatizing sound would separate other insertions and might even shake the cells in the organ of Corti loose from the basilar membrane, as in Lurie's[12] and Perlman's[17] observations.

If this concept of damage to the insertion is correct, then it would be probable that nerve deafness of the aged, so-called presbycusis, is in fact also a result of long-continued, oft-repeated, minimal acoustic trauma, possibly combined with degenerative changes resulting first in overstretching or actual tearing of the insertion of the outer margin of the tectorial membrane. Crowe, Guild and Polvogt[5] found further pathologic changes in definite sequence, which will be discussed in Study VI.

SUMMARY

A new concept of the pathology of presbycusis and acoustic trauma is presented. This pictures the earliest pathological change

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I can tell, the greatest intensity occurs just before the heel strikes the ground. Analyzing the movements during the cycle of the step, from known physical facts, it would be about as follows: As the body is raised up on the toe in the first half of the step, the otic capsule would rise with the rest of the head. The fluid within it, because of its inertia, would tend to lag. This would make a downward flow of fluid toward the lower window (the round window) during which there would be flow thru the helicotrema from the scala vestibuli into the scala tympani. As the body falls in the second half of the step, the otic capsule would also move downward in an accelerated velocity from the highest point in the step cycle to the lowest. Again the inertia of the fluid would cause it to lag, decreasing the pressure on the lower window and increasing the pressure on the upper window (the oval window). This would cause a flow through the helicotrema from the scala tympani into the scala vestibuli. The crowding of fluid into scala vestibuli would be greatest at the end of the period of flow and the point of greatest acceleration, which is just before the heel strikes the ground to stop the downward movement. At this point, the pressure within the scala vestibuli would be the greatest and, as nearly as I can tell in my own case, it is at this point that the tinnitus is the most intense.

Having made this logical explanation for the cyclic intensification of tinnitus in walking, one immediately begins to question its validity. Undoubtedly the physical forces described are there but are the magnitudes great enough to produce the observed effect? What about other fluids such as blood in the stria vascularis and the spinal fluid? Maybe they also do some swinging during walking. What causes the intensification of tinnitus when pressure is made on the side of the mandible or when the jaw is thrust forward or when pressure is made on top of the head? Is there enough distortion in the base of the skull and temporal bone from external stresses to cause pressure relations within the cochlea to alter in such a way as to influence tinnitus? If so, perhaps such distortion during walking causes pressure changes within the cochlea rather than inertia of the perilymph. What causes alleviation of tinnitus when the head is bent downward between the knees (in sitting)?

as damage to the insertion of the outer border of the tectorial membrane upon the cells of Hensen, causing loss of support here. Anatomic findings indicate that the tectorial membrane is held in position by the cells of Hensen and the border cells. If this support is lost, it is likely that tension is made upon the hairs of the hair cells. The accompanying tinnitus could be a result of this continuous traction. It is of course realized that there are other well recognized causes for tinnitus.

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XLVII

RECONSTRUCTION OF THE SUBGLOTTIC AIR PASSAGE

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Obstruction of the subglottic air passage presents the patient with the serious complication of asphyxia and the embarrassment of wearing a tracheotomy tube. Although it is frequently impossible to prevent the development of subglottic stenosis it is possible to correct the condition surgically. The six techniques applicable for various subglottic conditions assist in re-establishing and maintaining an adequate airway and functional vocal apparatus. Six illustrative cases are presented to identify these various techniques which have been used by many surgeons with modifications for over fifty years.

ETIOLOGY

The etiology of subglottic stenosis may be classified under the headings as: I. Congenital. II. Infection. III. New Growth. IV. Trauma.

I. Congenital.
   1. Cysts.
   2. Bands and webs.
   3. Congenital stenosis.
   4. Congenital atresia or absence.
   5. Congenital developmental abnormalities.

II. Infections.
   1. Tuberculosis.
   2. Diphtheria.
   3. Non-specific infection.
   4. Syphilis.
   5. Abscess formation.
   6. Post-irradiation necrosis with secondary infection.
   7. Scleroma.

III. New Growths.
   1. Malignant growths of the trachea or associated structures.

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a. Cancer of the trachea.
b. Sarcoma of the trachea.
c. Cancer of the thyroid gland and parathyroid glands.
d. Cancer of the larynx with subglottic extension.
e. Metastatic cancer of the deep cervical nodes.
f. Cancer of cervical esophagus.
g. Cancer of the hypopharynx.

2. Benign neoplasms.
   a. Chondroma.
   b. Adenoma.
   c. Mixed tumor.
   d. Fibroma.
   e. Enlarged thymus.
   f. Enlarged thyroid.
   g. Leukoplakia.
   h. Hemangioma.
   i. Branchial cyst.
   j. Dermoid cyst.
   k. Papillomata.

IV. Trauma.
   1. Crushing wounds of the trachea or larynx.
   2. Penetrating wounds of the trachea or larynx.
   3. Gunshot wounds of the trachea or larynx.
   4. Traumatic endoscopic procedures.
   5. Foreign bodies.
   6. Pressure from aneurysmal dilatations of the aorta or innominate vessels.
   7. High tracheostomy.

SYMPTOMS

The symptoms associated with subglottic obstruction quite naturally depend upon the etiology and degree of the obstruction. In minimal degrees of stenosis which are well compensated there may be no symptomatology whatsoever. The symptomatology may progress from this point and cover all degrees and variations of respiratory distress to the dramatic end point of asphyxia. Usually, however, by the time the doctor is consulted the patient has specific complaints related to the middle respiratory system, larynx, cervical esophagus or middle neck. These symptoms usually include hoarseness for any of the lesions affecting the production of sound. There may be difficulty in swallowing, obstruction to swallowing or spillage into the tracheal system, when the causative factor is located in the cervical

esophagus and hypopharynx. This may also be true in stenosis of the esophagus or esophageal-trachea fistula. The patient has frequently either a non-productive cough or cough associated with purulent or bloody sputum. The cough may be hacking in quality and persistent in character, frequently producing laryngeal spasm. Usually, however, the most important symptom in subglottic tracheal stenosis or obstruction is difficulty in respiration. It is progressive in character and frequently requires relief by the insertion of a low tracheotomy tube.

DIAGNOSIS

The diagnosis is usually accomplished by indirect laryngoscopy, direct laryngoscopy, lateral x-rays of the neck, tomographs of the neck and biopsy when indicated. If the patient's respirations are embarrassed a low tracheostomy should be performed before any other diagnostic techniques are carried out.

In adults indirect examination of the larynx and hypopharynx will frequently provide some of the strongest assistance in diagnosing hypopharyngeal, laryngeal and immediate subglottic lesions. Palpation of the neck is of assistance in establishing the presence of tumors in the thyroid gland and cervical lymph nodes. Roentgenographic and tomographic studies of the neck and chest will assist in establishing the presence of abnormal masses and contours in the various structures in these areas. Barium and lipiodol studies of the esophagus and hypopharynx are helpful. Direct laryngoscopy, bronchoscopy and esophagoscopy are usually necessary for an accurate evaluation of lesions at these sites. Direct endoscopic biopsy, aspiration biopsy or formal incisional biopsy in the neck is always necessary to establish the character of a neoplastic growth.

INDICATIONS

The indications for re-establishment of the subglottic airway concern themselves with the correction of the previously listed etiological factors. Those factors which either directly or indirectly cause a diminution in the subglottic air passages would in the vast majority of cases lend themselves to one of the six therapeutic methods of correction. Congenital defects, tumors and trauma to the subglottic area are corrected at the discretion of the surgeon and the pending urgency of the case. When the obstruction is secondary to specific or non-specific infection, this should be cured or arrested before surgery is attempted.

CLASSIFICATION OF THERAPY

The six therapeutic methods are classified as follows: I. Dilatation. II. Free graft only. III. Free graft and stent. IV. Direct ap-

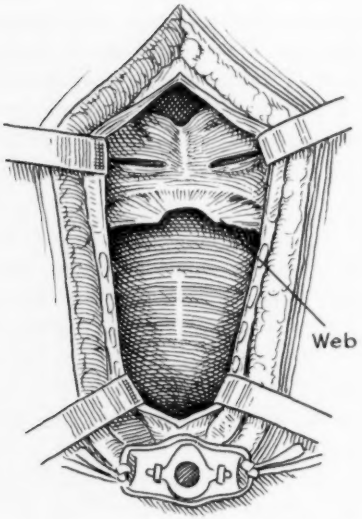

Fig. 1.—Web causing subglottic obstruction.

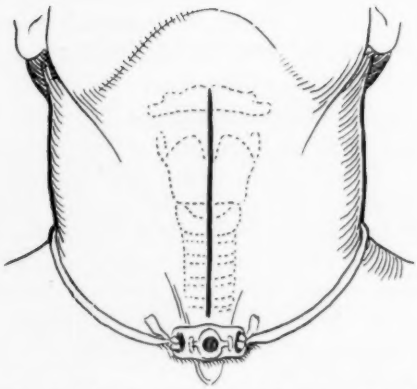

Fig. 2.—Incision for exposure of larynx and trachea.

proximation. V. Utilization of mucoperichondrial flap. VI. Reconstruction of the trachea.

I. "Dilatation" as a therapeutic technique is most applicable to subglottic obstructions which are constituted by thin to moderate webs. Massive amounts of scar tissue and extreme distortion of the trachea do not lend themselves to dilatation. These stretchings are accomplished by either intra-oral or retrograde use of hard or semi-hard dilators of various shapes and sizes repeated in a progressive manner over varying intervals of time. The treatment is usually protracted and inconvenient to the patient but is effective for cure of the thin to moderate web deformity (Case 1, Fig. 1).

II. "Free graft only" may be applied to the adult with a moderate sized subglottic constriction, obstruction or new growth, uncomplicated by excessive scar formation and in whom the cartilaginous framework of the trachea has remained intact. This technique is not advocated for infants or children because the contracture of the free graft postoperatively in small calibrated tracheas may defeat the purpose of the operation. The normal cartilaginous architecture of the trachea should be present in order to supply an appropriate framework for the skin graft. Tracheas that are absent, distorted or deficient are not satisfactory for the free graft only technique (Case 2, Fig. 2, 3).

This surgical technique is carried out through a longitudinal scar in the anterior part of the neck going beyond the limits of the subglottic lesion. All of the obstructing tissue is carefully removed and the raw wound created over the cartilaginous framework dressed with a thick split, free skin graft from a non-hair bearing area, carefully sutured into position with 4-0 catgut. This space is then packed with a modeled mold of foam rubber sponge wrapped in vaseline gauze. An attached heavy silk thread through the mouth permits extraction of the sponge within a period of approximately ten days without the necessity of reopening the neck wound. Foam rubber sponge is a desirable type of dressing material because it molds itself to the irregular contour of the trachea and creates a desirable amount of pressure in approximating the skin graft to the tracheal bed. Wrapping it with vaseline gauze facilitates its removal at a later date by maintaining a lubricating film between the dressing and the skin graft thus permitting withdrawal of the packing without dislodgment of the skin graft.

III. "Free skin graft and stent." The principle of incorporation of the semi-permanent stent in the reconstruction adds resistance to the natural contractile forces and assists in producing an adequate

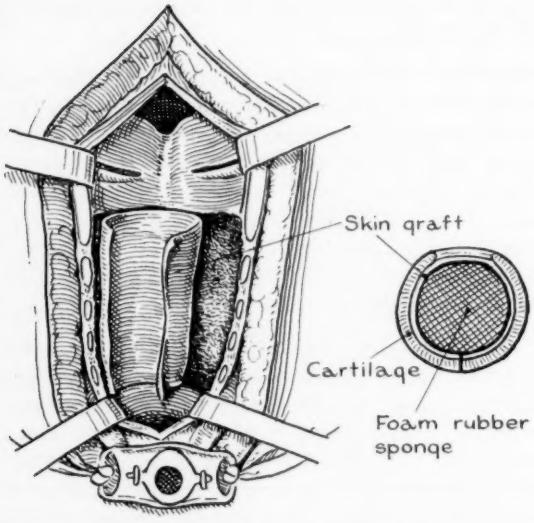

Fig. 3.—Skin graft held in trachea by foam rubber sponge which is removed through the mouth after one week.

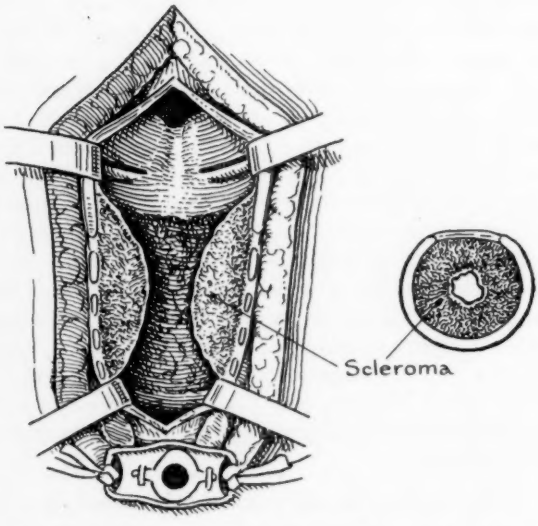

Fig. 4.—Obstruction to the subglottic area by extensive scleroma.

and normal air passage. The reconstruction of the subglottic area in an infant or child not only requires removal of the deformity but the maintenance of an airway with a stent beyond the time when scar contracture might defeat the purpose of the procedure. The principle of the stent is also required when the tracheal framework is excessively deficient, absent, markedly distorted or obstructed (Case 3, Fig. 4, 5).

This technique is carried out in a manner similar to the technique employed for free graft only in that a longitudinal wound is made in the anterior part of the neck going somewhat beyond the obstructing tissue in the subglottic area. All of the obstructing tissues are removed. If it is possible to salvage the tracheal cartilages and re-assemble them in their normal position that should be done. After the thick split, free skin graft is sewed into position with 4-0 catgut the wound is then dressed with a pressure dressing which will remain in situ for three to ten months. Again foam rubber sponge offers an ideal type of dressing material in that it creates a desirable amount of pressure against the contractile forces of the wound. It should be placed in a heavy, elongated rubber balloon to prevent its absorbing and retaining secretions. It is not desirable under these circumstances to bring a string out through the mouth because of the associated prolonged inconvencience. The indwelling mold should be directly sewed to the wound by deep silk sutures. As time goes on the mold has a tendency to become freer in the wound as a result of its dilatory action in the subglottic area and also the result of the many movements of the head associated with swallowing, speaking and moving the neck. One must take care that this mold does not slip into the low part of the trachea, where it could cause asphyxia when the tracheostomy tube is out for cleaning or inspection. To overcome some of the inconveniences associated with an elastic rubber dressing in the subglottic area plastic molds have been made which are inserted into the wound several weeks after the skin grafting technique. They have the advantages of being cleaner, less affected by tissue juices and are more easily secured in position. They have the disadvantages, however, of not conforming in as smug a manner to the contour of the trachea. After the desired period of time, which may comprise three to twelve months, depending upon the amount of scar contracture which the mold must prevent, the neck wound is partially re-opened and the implant removed. After approximation of the wound and corking of the tube for a safe period of two or three weeks one may consider decannulization.

IV. "Direct approximation of the trachea." Where there is a localized obstruction in the subglottic area involving only one or two

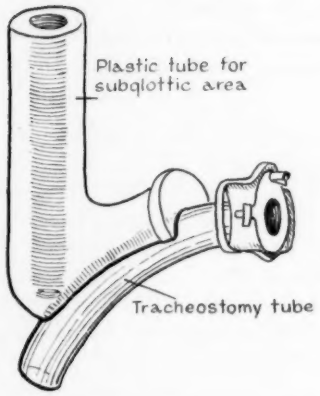

Fig. 5.—Acrylic mold to prevent post-operative subglottic contraction.

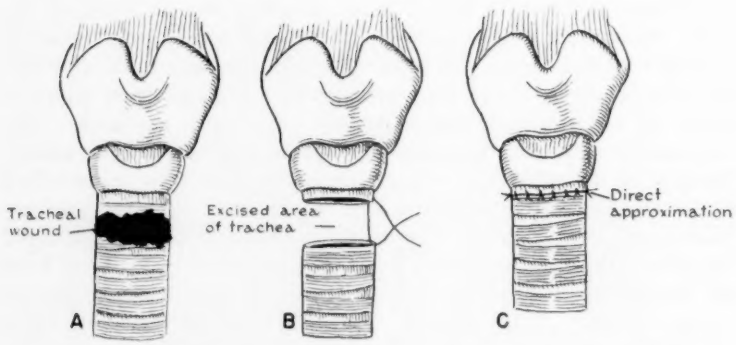

Fig. 6.—Wound of trachea repaired by minimal debridement and direct approximation.

of the cartilaginous rings associated with moderate distortion, web formation with or without fistulization, one can immediately reconstitute the subglottic airway passage after the appropriate excision of the obstructing mass by literating the trachea superiorly and inferiorly and approximating the two segments. The same principle may be applied to the excision of the cricoid cartilage by approximating the first tracheal cartilage to the inner surface of the thyroid cartilage. The trachea is unusually flexible and may be stretched somewhat in the fashion of opening an accordian. This stretching, however, does not permit the incision of more than two cartilage rings. The cut edges of the trachea are re-approximated with individual sutures of 2-0 chromic catgut. Care should be taken in this procedure to preserve the integrity of the recurrent laryngeal nerves. This procedure has the advantage over grafting in that there is no foreign material inserted into the air passage. It is perhaps a procedure that should be used more often in surgery on the trachea (Case 4, Fig. 6).

V. "Utilization of mucoperichondrial flaps." Benign tumors and obstructions of the cricoid and tracheal cartilages which have caused serious impairment to the airway system may be cured, when the mucosa is intact, by a subperichondrial excision of the tumor and the re-positioning of the mucoperichondrial flap of the trachea over the wound. This procedure has the advantage of immediately reconstituting the mucosal lining of the trachea without the need of skin grafting or implantation of a semi-permanent mold. After the mucoperichondrial flaps are carefully positioned into the bed of the trachea, a pressure dressing of foam rubber sponge wrapped with vaseline gauze is applied over the flap. A heavy string is brought out through the mouth so that this packing may be removed at the end of a period of one week without the necessity of re-opening the neck wound (Case 5, Fig. 7).

VI. "Reconstruction of the subglottic area." When a large section of the trachea and associated tissues have been destroyed or removed and it is impossible to apply any of the above surgical principles, one must consider first the immediate problem of continued maintenance of the airway and secondly the permanent reconstruction of the airway passage. Pathological conditions necessitating these considerations are usually associated with a radical excision of malignant tumors in these areas but may also encompass large benign tumors or extensive trauma and congenital defects. Immediate maintenance of the airway system has been successfully carried out by the use of inert tubes consisting of plastic, metal and rubber

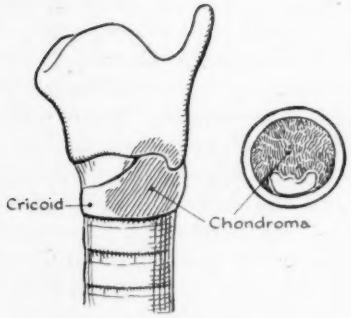

Fig. 7.—Large cricoid chondroma showing airway obstruction and intact epithelium.

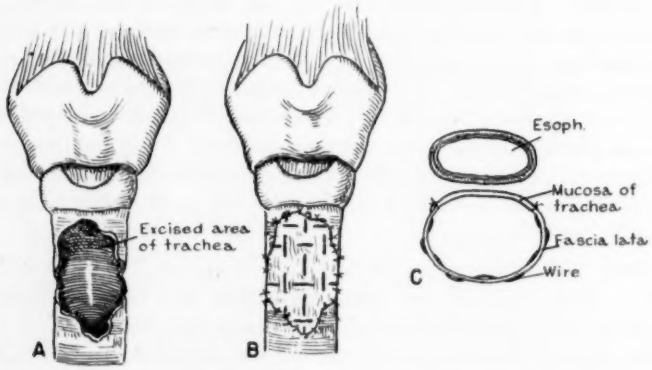

Fig. 8.—Reconstruction of tracheal defect by tantalum wire woven through fascial graft.

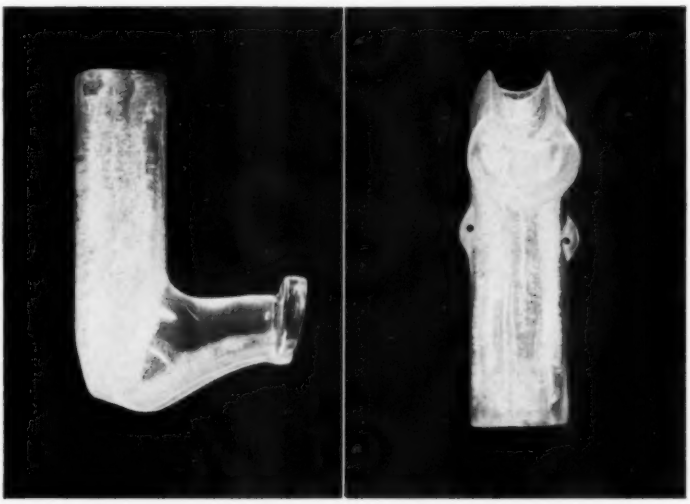

Fig. 9.—Case 3a, Acrylic mold for infraglottic area. Case 3b, Acrylic mold for infraglottic area.

composition. These, however, are not desirable as permanent embodiments of the trachea. Homografts of trachea, fascia and cartilage have not been totally successful or satisfactory. Autografts alone or in conjunction with an inert substance have proven successful. The immediate reconstruction of these large tracheal defects by the use of either fascia or dermis supported in the wound by a tantalum or stainless steel wire network has met with success (Case 6, Fig. 8). The basic accomplishment here is one of providing a substantial circular architecture over which the respiratory mucous membrane may regenerate. This mucosa fortunately has the capacity of rapid and total regeneration. The transplantation of autogenous cartilage may also assist in reconstituting the tubular form of the trachea and preventing collapse or excessive cicatricial contracture. The tracheal or bronchial graft has been used successfully in some instances where it has been necessary to resect a portion of the lung, bronchi and trachea with a salvaging of some of the bronchial elements from the surgical speciment and re-implantation into the wound in an effort to reconstruct and maintain the air passage. This principle may be applied in the trachea by the acquisition of smaller sections of tracheal substance (one to two rings) for transplantation to a larger deficient area.

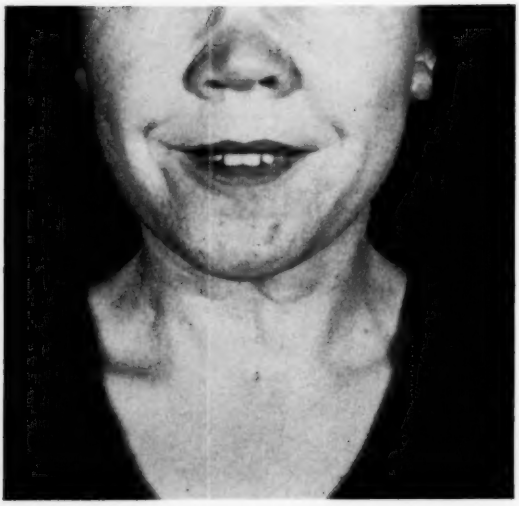

Fig. 10.—Case 3c, Patient one year after decannulization.

CASE 1.—Mr. R. C. had had a resection of multiple papillomata of the larynx and subglottic areas by direct thyrotomy and cricoidotomy in 1937 at the age of 12 years. His airway had been borderline for many years following this procedure, with limitation of excessive physical activity. In 1946, he developed a severe laryngitis and tracheitis and it was necessary to insert a low tracheostomy tube. On direct and indirect examination in September of 1946 a thin web was revealed in the immediate subglottic area, arising anteriorly and extending toward the posterior part of the cricoid in a bilateral crescent-shaped fashion. This thin web had reduced the subglottic air passage by approximately 60%. It was progressively dilated by the oral and retrograde methods twice a week for a period of three months with graduated dilators. The dilators were allowed to remain in position for a period of ten to fifteen minutes upon each technique. Approximately 80% of the web disappeared and the patient was decannulated and has been asymptomatic since that time. There has been no tendency for the web to recur.

CASE 2.—Mrs. M. G., aged 49, revealed that she had had a tendency to crusting and scab formation in her nasal cavity all of her life. Approximately four and a half years ago it had become difficult for her to breath and a tracheostomy tube was inserted. She

was told at that time that there was subglottic swelling and emeda and possibly scar formation. This area was treated with irradiation (amount not known) and after a period of nine months she was improved although she states that she never had quite enough breathing space.

In January 1952, her airway became markedly obstructed and examination at that time revealed extensive crusting and atrophic rhinitis in the nasal cavity with a marked purulent postnasal drip. The vocal cords moved moderately well. The arytenoids were not displaced. Immediately beneath the vocal cords were two prominent subglottic scar tissue masses which were firm and immobile and diminished the airway to approximately 30% or normal capacity. Wasserman examination was negative and biopsy of this subglottic mass revealed dense fibrous tissue.

A longitudinal incision was made through the thyroid, cricoid and upper four tracheal rings. The entire scar was carefully removed from the undersurface of the vocal cords down to the third tracheal ring. The wound was repaired by the implantation of a thick-split skin graft held in position with foam rubber sponge and vaselinized gauze. The cartilages and neck wounds were snuggly approximated over this inlay graft and a low tracheostomy tube was inserted. The postoperative course was complicated by the constant formation of lower tracheal and bronchial scabs, encrustations and casts. This dangerous complication was treated by (1) high humidity in an oxygen tent, (2) potassium iodide and ammonium chloride by mouth, (3) frequent flushings and aspirations of the tracheal and bronchial tree with sterile saline solution and (4) multiple bedside bronchoscopies (as many as six in one day).

On the eighth posoperative day the foam rubber sponge and vaselinized gauze were removed through the neck wound. The airway was adequate and the lower respiratory encrustations disappeared as soon as the patient began to breath normally through the upper respiratory passages. Since that time the patient's physical activity is not restricted in any manner.

CASE 3.—Mrs. C. S., age 35, revealed that twelve years ago she was admitted to the hospital in acute respiratory distress and that an emergency tracheostomy was performed. Later a laryngofissure and splitting of the trachea and cricoid cartilages was performed. The entire glottic and subglottic areas from slightly above the larynx down to approximately the fifth tracheal ring were completely occluded with a vegetative growth of papillomatous tissue. This papillomatous mass was removed in toto and although the patient's im-

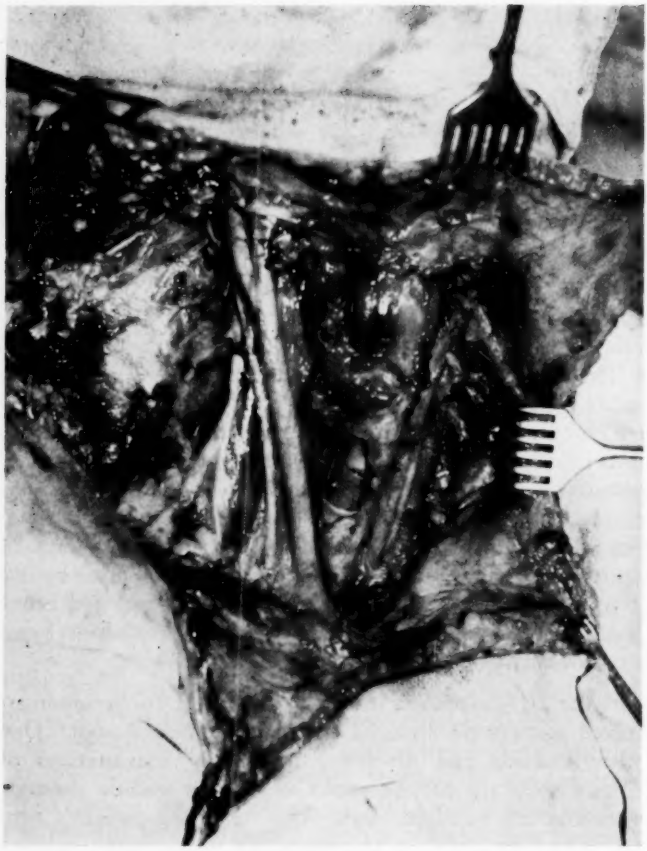

Fig. 11.—Case 6a, Operative area revealing bilateral radical neck dissection, total thyroid- and parathyroidectomy and large tracheal defect.

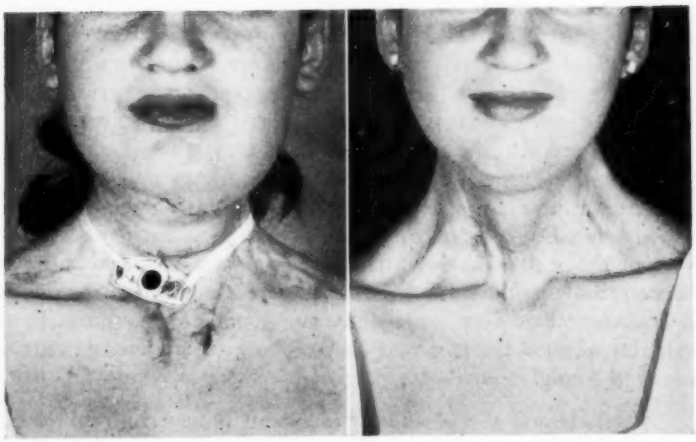

Fig. 12.—Case 6b, Patient three weeks postoperative. Case 6c, Patient one and a half years postoperative.

mediate airway was improved, she subsequently became dependent upon her tracheostomy tube. For the next year and a half she was dilated by the retrograde and oral approaches with only slight improvement. She never reached the state during this period of dilatations where she could function with her tracheostomy tube corked. In 1946 the thyroid and cricoid cartilages and upper sixth tracheal rings were opened and the scar tissue in these areas excised. The raw bed was covered with a Thiersch graft which extended from just beneath the vocal cords to the sixth tracheal ring. The patient was markedly improved after this technique and was capable of performing light housework and sleeping with the tube corked for a period of approximately six months. She then began to recognize constriction of her airway and although she could breath with the tracheostomy tube corked, it was an effort.

In January 1951 this patient came under our observation. Examination of the larynx revealed a rather infantile larynx with fixation of both cords and both arytenoids. The laryngeal airway was approximately one millimeter in width. The area immediately beneath the cords was compressed by scar tissue to approximately a diameter of one millimeter. The tissue was firm and resembled scar tissue. There was a foul odor to the breath coming from stagnation and epithelial debris. The solid mass of scar tissue and old skin graft

extended down to the fifth tracheal ring. In January 1951 the thyroid, cricoid, tracheal cartilages were opened through the old scar and the old skin graft and heavy formation of scar tissue were surgically excised. The scar on the undersurface of the vocal cords and about the vocalis muscle itself was also excised. A previously prepared acrylic mold about which was wrapped a thick-split skin graft was then placed into the raw wound. The acrylic mold and free skin graft extended from the sixth tracheal ring into the supra-glottic areas. It was possible to readily see the tip of this acrylic mold above the vocal cords by the indirect examination. The cartilages and soft tissues of the neck were then approximated about the mold and a low tracheostomy tube inserted. The postoperative course was uneventful and there was very little difficulty in swallowing normally by mouth in spite of the fact that the acrylic mold abutted against the base of the epiglottis.

After a period of one year the acrylic mold was removed through the original incision in the neck. The tracheostomy tube was removed at the same time and the patient encouraged to breathe through the newly created channel by occluding the tracheastrome with an adhesive bandage. In a month the tracheastome had diminished to approximately 20% of its original size and was then closed. The patient's airway since that time has been adequate for all types of physical activity. The arytenoids do not move and the voice is only of fair quality, resembling a strong hoarse whisper.

CASE 4.—Mr. J. N., age 28, had been wounded in the trachea by shrapnel in World War II. A fistula was present in the anterior neck and a web of scar tissue had formed involving the second and third tracheal cartilages with a diminution of the airway to approximately 20% of its original diameter. The web was primarily anterior in position and had a broad base.

In April 1944, the trachea was exposed through a longitudinal incision in the neck from the cricoid cartilage to the seventh tracheal ring. A scarred and twisted second and third tracheal ring along with their associated web and fistula were surgically excised causing a deficiency in the trachea. This defect was corrected by liberating the tracheal inferiorly and doing an end-to-end approximation of the first to the fourth tracheal ring with 00 chromic catgut. The head was maintained in a flexed position postoperatively. The tracheostomy tube was removed at the end of the third postoperative week and the patient had an uneventful convalescence.

CASE 5.—Mr. C. O., age 62, related that he had become progressively hoarse over the past six months and that this was associated

with progressively increased difficulty in breathing. He had developed an irritating, non-productive cough which would at times cause spasm of the throat that was greatly alarming to the patient.

Indirect and direct laryngoscopy revealed a firm, protuberant mass with a very broad base arising from the posterior surface of the cricoid cartilage in the immediate subglottic area diminishing the airway to approximately three millimeters in diameter. The mucus membrane over this tumorous mass was intact. The vocal cords were injected and relaxed but mobility was good. Biopsy of the tumorous mass revealed a chondroma of the cricoid cartilage.

In March 1951, the cricoid and first tracheal cartilages were split anteriorly. A subperichondrial dissection of the cricoid was carried out exposing the entire cricoid cartilage and chondroma. The chondroma comprised approximately 90% of the cartilage so it was decided to remove the entire cricoid. This wound was repaired by carefully suturing the mucoperichondrium over the cricoid bed and maintaining it in position with a foam rubber sponge dressing wrapped with vaselinized gauze. This packing was maintained in position by the snug closure of the peri-cricoid and tracheal tissues about it. Further security was obtained by bringing a piece of heavy silk out through the mouth and another one out through the tracheal opening. On the eighth postoperative day the inferior piece of silk in the tracheal opening was cut and the foam rubber and vaselinized pack gently delivered through the mouth. This was done without any unusual event in the patient's room, thus eliminating an additional surgical technique.

The patient was breathing normally in three weeks. The airway is adequate, the vocal cords move well, although they are somewhat relaxed and the voice is baritone in quality. Subsequent examinations of the cricoid area reveal it to appear normal. There is no regrowth of the chondroma and the patient's activities are not restricted.

CASE 6.—Mrs. M. M., age 22, reported in September 1950 with a biopsy diagnosis of adenocarcinoma of the thyroid. It had invaded the first three tracheal rings and the tracheo-esophageal sulcus bilaterally and had metastasized to both lateral necks. A total thyroidectomy, parathyroidectomy, resection of the involved trachea and bilateral radical neck dissection was performed in September 1950. Both recurrent laryngeal nerves were sacrificed and a large tracheostome at the level of the excised trachea was created. Three months following this operation, the left arytenoid was laterally displaced in order to gain an adequate laryngeal air passage and the defect in the

trachea corrected with a tantalum wire mesh and fascial graft covered by appropriate skin flaps. The patient's voice has the quality of a strong whisper, but her airway is adequate for all activity. She has had no recurrence of her tumor.

SUMMARY

1. A classification of subglottic stenosis is made.

2. Six methods of treatment are described for the various types of subglottic deformities.

3. Six case reports are presented to illustrate the various techniques.

139 EAST 36TH STREET.

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XLVIII

THE CONDITION OF FLUID IN THE MIDDLE EAR

FACTORS INFLUENCING THE PROGNOSIS IN 56 CHILDREN

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The object of this study is to present the conflicting findings in 56 cases of fluid in the middle ear* in children noting in particular the factors which seemed to be related to an unfavorable outcome. Observations were made on the viscosity and microscopic findings in the fluid taken from the middle ear, and their influence on the ultimate result of the case. Eosinophils and bacteria were found in this fluid (twice in combination) and apparently had a bearing on the subsequent course of the case. In the experience of the writer, certain cases of fluid in the middle ear run a protracted or relapsing course in spite of all efforts at a cure. Other cases, however, seemed to respond with little or no treatment.

In each of the 56 cases on which this study is based, the tonsils and the adenoids (or the adenoids alone) were removed, following which myringotomy was performed and the fluid evacuated from the affected middle ear.

This work was done in the period from 1947 to 1951. All cases were in children of less than 10 years of age. In approximately three-fourths of these cases (43), the tonsils and adenoids were hypertrophied and infected. Each case was followed for at least two months; approximately half were followed for six months, and two cases were observed for four years.

The results have been tabulated as recoveries and non-recoveries. Recovery is defined as a resolution of the process in the middle ear without relapse and with a return of the hearing to normal. By non-recovery it is meant that the condition continued as before treatment or that it recurred.

Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1952.

*Secretory otitis media, catarrhal otitis media, serous otitis media, otitis media serosa, sero-mucus middle ear catarrh, exudative middle ear catarrh, catarrh of the tympanic cavity and eustachian tube, non-suppurative otitis media, hydrops of the middle ear, tubo-tympanic catarrh.

These 56 cases fell naturally into three groups. In the first group the fluid in the middle ear was of a thin, serous type and the eustachian tube was seen to be normally patent as tested by the procedure of tympano-tubal lavage. Tympano-tubal lavage is a procedure described by Brown[2] in which myringotomy is performed (if there is no perforation in the ear drum) and by the use of positive pressure in the external auditory canal, the middle ear and eustachian tube are irrigated into the nasopharynx with air or solution. In the second group the fluid in the middle ear was of a thick mucoid type, and the eustachian tube was not normally patent as tested by the above procedure. In the third group one ear contained thin fluid as in the first group, whereas the other ear contained thick fluid as in the second group.

After tonsillectomy and adenoidectomy were completed, and hemostasis had been accomplished, an opening was made in the anterior inferior quadrant of the membrana tympani in the affected ear. In order to test the patency of the eustachian tube, slight positive pressure was produced in the external auditory canal with the Siegle-type otoscope. As much fluid as possible was then evacuated from the middle ear into the external auditory canal. This was done by negative pressure (preferably made by the use of the Siegle-type otoscope).

Preparations for microscopic study were obtained from the fresh fluid taken from the middle ear, and the smears were made by the operator directly from the ear to the slide. Smears were prepared also from the "plug" when it was present. The Hartman-type ear forceps were preferred to a cotton swab in handling the material and preparing the smear. Care was taken to be gentle since eosinophiles rupture easily with consequent scattering of the granules. These smears were stained by the Hansel separate technic. When bacteria were found, additional smears were stained by the Gram method.

In the first group (in which the fluid in the middle ear was of thin consistency) the hearing loss was moderate. When alternating positive and negative pressure was applied by the use of a Siegle-type magnifying otoscope, the ear drum moved freely. The handle of the malleus was normal in appearance. The drum was thin, semi-transparent, and fluid could be seen through it. Frequently the fluid contained air bubbles and at times a distinct fluid level was seen. Rarely was the middle ear completely filled with fluid, and inflation by Politzer's method could be performed without difficulty.

TABLE I.

MICROSCOPIC FINDINGS IN THE THIN FLUID FROM THE MIDDLE EAR IN 20 CASES.

	RECOVERIES	NON-RECOVERIES
Eosinophils	0	0
Bacteria	0	0
Eosinophils and Bacteria	0	0
Neutrophils Only	5	0
No Microscopic Findings	12	3
TOTAL	17	3

In the second group (in which the fluid in the middle ear was thick and viscid) the hearing loss was greater and the history of symptoms was usually longer than in the first group. The handle of the malleus was chalky white in color and stood out prominently. The drum was thick and opaque and it was not possible to see fluid through it. When alternating positive and negative pressure, however, was applied to the drum by the use of the Siegle-type magnifying otoscope, it was apparent by the limitation of motion that there was thick fluid medial to the drum. Inflation by Politzer's method was difficult, even impossible at times. After myringotomy, a thick, sticky, ropy, amber-colored material was seen within the middle ear cavity. The eustachian tube portion of the middle ear contained an even thicker substance which mechanically blocked the eustachian tube. This substance is evidently what Politzer referred to as a "mucous plug." Often it required several minutes of manipulation with the Siegle-type otoscope or with a water-connected suction tip to aspirate all of this material through the myringotomy incision into the external auditory canal. Until the thicker substance in the eustachian tube portion of the middle ear was freed from its position, it was usually impossible to irrigate the eustachian tube by tympanotubal lavage. After this substance was removed, however, tympanotubal lavage with air or saline was carried out easily (except in three instances). The reason for the difficulty in these three exceptions is not known; two of them were classified as recoveries.

Not only were the two groups distinguished by the degree of viscosity of the fluid in the middle ear, but they were also distinguished by the microscopic findings in the fluid.

In group one, smears made from the thin fluid frequently contained no cellular elements. In a few cases neutrophilic leucocytes were found, but no eosinophils or bacteria were found in smears made from fluid of thin consistency (Table I).

In group two, where smears were made from the viscid material, microscopic findings were different. Neutrophilic leucocytes were found in large numbers, and in many instances they were the only type of cell found. But in other cases either bacteria or eosinophils were found (Tables II and III). In smears made from the "plug" itself, a larger number of bacteria and eosinophils were present than in smears made from the first fluid obtained from the middle ear. The percentage of eosinophils found in the fluid in cases tabulated as containing this cell, was from approximately five to ten per cent, except in one case in which it was approximately 100 per cent.

A varied group of bacteria was found in these smears. A large gram-positive diplococcus was seen in four cases; this same organism was found in combination with a mixed flora of other bacteria in three cases; gram-negative rods and diptheroids were observed in two cases; and the type of bacteria was not classified in two cases. All cultures were either sterile or grew what was considered to be a contaminant.

One case in this series stands alone and was not included in the tables or the comparative figures in the summary. In this individual, bacteria were found in the fluid of the right middle ear, and eosinophils in the fluid of the left middle ear. The ear which contained bacteria resolved with apparent cure, but the ear which contained eosinophils did not improve with treatment.

COMMENT

The finding of an appreciable number of eosinophils in the fluid obtained from the middle ear is taken as presumptive evidence of an allergic process. In six of the nine cases in this series where eosinophils were found, there was definite allergic rhinitis or asthma. The absence of eosinophils apparently does not rule out a diagnosis of allergy. In five cases in which no eosinophils were found there was definite allergic rhinitis or asthma. On the other hand, in three cases in which eosinophils were observed, no other evidence of aan allergic process was apparent.

TABLE II.

MICROSCOPIC FINDINGS IN THE THICK FLUID FROM THE MIDDLE EAR IN 20 CASES.

	RECOVERIES	NON-RECOVERIES
Eosinophils	1	3
Bacteria	4	1
Eosinophils and Bacteria	1	1
Neutrophils Only	3	4
No Microscopic Findings	2	0
TOTAL	11	9

TABLE III.

MICROSCOPIC FINDINGS IN 15 CASES IN WHICH ONE EAR CONTAINED THIN FLUID AND THE OTHER CONTAINED THICK FLUID.

	RECOVERIES		NON-RECOVERIES	
	THIN	THICK	THIN	THICK
Eosinophils	0	1	0	2
Bacteria	0	3	0	1
Eosinophils and Bacteria	0	0	0	0
Neutrophils Only	3	3	1	3
No Microscopic Findings	9	1	2	1
TOTAL	12	8	3	7

In three of the cases in which eosinophils were found and allergy was present, an investigation of allergy was made and treatment was given. Sensitivity to a specific allergen either was not found or was not found to be important. These cases were placed on a regime which consisted of the injection of various doses of dilute dust extract and other vaccines, the oral administration of antihistamines, desensitization to seasonal pollens, precautions for dust avoidance, and elimination diets. None of these had any obvious effect on the process taking place in the middle ear.

The presence of bacteria in the fluid does not necessarily indicate that a bacterial infection was the primary cause of the process in the middle ear. It is considered more likely to be a low-grade secondary infection, possibly of the fluid itself (as suggested by Politzer). Five cases were observed for a period of time before the operation. In these cases there was thin fluid present in the middle ear with minor symptoms. However, with an acute upper respiratory infection, or for no apparent reason, the picture changed. The thin fluid became thick, the ear drum became swollen and slightly hyperemic, and the hearing decreased considerably. Occasionally there was slight earache. This apparently inflammatory process usually subsided after antibiotic therapy, and the ear returned to its previous state as evidenced by the presence of thin fluid in the middle ear.

Three patients classified as non-recoveries were followed postoperatively for a period of several months. On several occasions evidence of secondary infection similar to that described above was seen. Sometimes with the use of antibiotics the process appeared to resolve completely, but frequently it recurred after treatment was discontinued.

Aureomycin was found to be the most effective drug used in these particular cases. Terramycin and chloramphenicol were found to be next in effectiveness; the least effective was penicillin. Streptomycin and the sulfonamides were not used often enough to allow the observer to formulate an opinion concerning their effectiveness.

The presence of both eosinophils and bacteria in the fluid from the same ear in two cases suggested the possibility that both an infectious and allergic process were taking place at the same time. In each of these cases, definite allergic rhinitis was noted, and in one, asthma was proved.

Roentgenograms of the mastoid processes were made in eleven cases classified as non-recoveries. Nine of these eleven showed sufficient changes to indicate that a process similar to that observed in

the middle ear was taking place in the mastoid cells. In no case, however, was destruction sufficient to warrant surgical interference.

During the period in which this study was made, an additional related case was seen which was so unusual that it deserves brief mention. A 38-year-old male had a severe case of fluid in the middle ear and the largest adenoids ever seen by the writer: the nasopharynx was almost completely occluded. The duration of symptoms had been brief and a malignant tumor was suspected. The adenoidal mass was removed and myringotomy was performed with evacuation of the fluid from the middle ear. The pathologist reported "chronic adenoiditis." A smear of the fluid showed only neutrophils. Neither the adenoids nor the fluid in the middle ear has recurred after four years.

REVIEW OF THE LITERATURE

In view of the current interest in the condition of fluid in the middle ear, it is surprising to find how well the process was understood by practitioners in the 19th century. In 1817, Wright[25] recognized this condition and believed that the eustachian tube was blocked by mucus caused "from the effect of colds and a variety of undeterminate causes." He noted that perforation of the membrana tympani gave temporary relief of symptoms, but that such a procedure was not always of permanent value; in fact, it might even be harmful.

In 1821, Itard[12] was familiar with the process and thought it was caused by tumors of the nasopharynx, enlarged adenoids, catarrhal or mucous engorgement of the eustachian tube, or stenosis of the eustachian tube by adhesions. He treated the condition by myringotomy followed by inflation with the Valsalva maneuver, sometimes using camphorated tobacco smoke. He performed adenoidectomy in some cases and found the procedure to be a "little difficult." Blegvad[1] mentions a later work of Itard in which he advocated the use of his eustachian catheter in the treatment of the process.

Toynbee,[23] prior to 1860, believed that catheterization of the eustachian tube was harmful in these cases, and preferred the use of an "air douche" of the middle ear similar to Politzer's later method of inflation. This may well have been the beginning of a century of controversy. Toynbee also attempted to treat this condition by making a permanent perforation in the membrana tympani and described the difficulty in keeping the perforation open. He even devised a syringe for aspirating the fluid through the ear drum, although he used it in only a few cases.

In 1868, Hinton[7] gave four detailed case histories of the accumulation of mucus in the tympanum. The findings and the results of treatment in these cases were similar in many respects to those observed by this writer. In speaking of the common occurrence of the condition, Hinton stated that "it is only quite recently that their great frequency has been understood."

Politzer's section on "Catarrhs of the Middle Ear" in his textbook[17] (1878) is one of the most authoritative and comprehensive treatises on the condition of fluid in the middle ear which exists in the literature. This is attested to by the frequency with which his findings are quoted by widely-known authorities on the subject.

Politzer[17] was the first, evidently, to note a relation between the viscosity of the fluid and the prognosis. He stated that the quality of the fluid had an important influence on the course of this condition. He thought that "serous secretions" in the middle ear were absorbed more rapidly than "tough, syrupy, adhesive, mucous masses." Hoople[8] has also referred to this finding and mentioned that serous fluid might change to mucous fluid. Hoople also found that in some cases there was a mixture of the two types of fluid in the same ear. Hantman[6] pointed out that in bilateral involvement of the middle ear, one cavity could contain serous-type fluid and the other the mucous-type.

Hoople considers the fluid of a thin serous character to be a transudate and the mucous fluid to be an exudate, the former having a high protein content and the latter a low protein content. Robison and Nicholas[19] believe that as an "effusion" remains in the middle ear for a protracted period of time, the protein content increases and its colloidal osmotic pressure increases until it is higher than the colloidal osmotic pressure of the plasma. This precludes the absorption of the effused fluid by the plasma, and may act as an osmotic wick and draw in more fluid from the plasma.

Lowe[16] has reported the similarity between the fluid in the middle ear and the blood serum. In 15 cases in which the fluid was aspirated from the middle ear, he found that the blood group could be easily demonstrated by examination of this "exudate." This could not be done with the discharge of purulent otitis media. In a patient with vasomotor rhinitis and "secretory catarrh" of the middle ear, he compared the nasal secretion with the fluid in the middle ear. He found that the nasal secretion had a lower protein content and that the blood group could not be determined from it, whereas the fluid in the middle ear had a higher protein content and the blood group could be easily determined. Dohlman[4] has compared the similarity

between the thick fluid in the middle ear and the thick mucoid bronchial secretion in asthma.

Most observers have considered the fluid to be sterile (Scheibe,[20] Robison,[18] Dohlman,[4] Hoople and Blaisdell,[10, 11] Shahinian,[21] and others), but there is evidence to the contrary. Blegvad,[1] in 1932, reported a bacteriologic investigation of the fluid in "secretory" or "catarrhal otitis media." Out of 53 cases, bacteria were found in 26, either in the smear or in the culture made from this fluid. In 7 cases, bacteria were observed in the smears but did not grow in the cultures. In 11 cases, bacteria were discovered in the cultures but were not found in the smears. In the latter instance he did not state whether he had considered the possibility of contamination. He believed that the low virulence of the organism involved might account for the sterility of the culture when the corresponding smear presented bacteria.

Forschner,[5] in 1926, inoculated guinea pigs with fluid aspirated from the middle ear and made cultures as well. From the results of this and other studies he concluded that the bacteriologic picture was obscure. Robison and Anigstein have also inoculated guinea pigs, some of which were virus susceptible, with the fluid aspirated from the middle ear in 17 cases. The animals showed no ill effects. Tobey,[22] on the other hand, has advanced an interesting theory that "serous otitis media" is caused by an avirulent virus infection.

Dohlman, in 1943, presented microscopic evidence that this condition is sometimes an allergic process. He stated that although cellular elements were frequently scarce, eosinophils were sometimes found in large numbers. Robison, however, did not find eosinophils in over 100 smears made from fluid aspirated from the middle ear. Hotchkiss[26] considered that even in definitely allergic cases the eosinophilic count in the fluid of the middle ear is not dependable. Allergy, as an important etiologic agent in this condition, has also been discussed by Shahinian, Hoople, Williams,[24] and Jordan.[14]

It has become well established that mastoid involvement may occur in this process and is not a favorable prognostic sign. Hoople considered that in some cases the rapid reappearance of the fluid after removal, was due to mastoid involvement. Robison,[18] in his classification of different types of this condition, describes one as "acute catarrhal mastoiditis with effusion." Jervey,[13] Lawson,[15] Cody[3] and Tobey[22] have all reported cases of mastoiditis requiring surgery, as a complication of fluid in the middle ear.

SUMMARY AND CONCLUSIONS

Presented here are the findings in 56 cases of fluid in the middle ear of children, in which tonsillectomy and adenoidectomy were performed with myringotomy and evacuation of the fluid.

The viscosity and the microscopic findings in the fluid are correlated with the outcome in these cases. Cases in which the fluid was thick and mucoid were found to be more likely to constitute a difficult problem than those in which the fluid was thin. This finding confirms the previous observations of Politzer and Hoople.

Eosinophils or bacteria were found in 18 of the 35 cases in which thick fluid was present. The thin fluid showed only neutrophils, and in the majority of cases nothing at all.

Eosinophils were found more frequently in cases classified as non-recoveries. Conversely, bacteria were found more frequently in cases classified as recoveries.

The presence of bacteria in 11 cases was considered as evidence of a low-grade secondary infection. This was substantiated by additional clinical evidence.

The occurrence of both eosinophils and bacteria in the same ears in two cases suggested the possibility of a co-existing allergic and infectious process.

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XLIX

IS THERE A SPECIAL NUTRITIVE CELLULAR SYSTEM AROUND THE HAIR CELLS IN THE ORGAN OF CORTI?

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Most surface epithelia have in their immediate neighborhood a rich capillary system for nutrition. In the organ of Corti there is no such capillary system and the only adjacent capillaries are found on the tympanic side of the basilar membrane below the tunnel of Corti, in the *vas spirale*. For nutrition and oxygenation during the stimulation of the organ of hearing it seems necessary that the sensory elements, the hair cells, must have a highly specialized nutritive aid system. Such a system has hitherto not been described although most observers regard the Deiter cells as such a structure. It has also been suggested that the vascular stria composes the most important source in the production of the endolymph and thus also the most important nutritive source for the hair cells. By intermediation of the supporting structures of the organ of Corti the hair cells would then be nourished. There has, however, not been described any type of resorptive organs on the Deiter cells or in the organ of Corti.

By using modern electron microscopic methods for the study of the organ of Corti it is possible to achieve a far better understanding of the structural elements in this organ than has hitherto been possible with light microscopy. We have observed, that the Hensen cells on their surfaces have a rich system of very minute thin-walled processes with many similarities to other resorptive structures and in the following a short description is given of these structures.

The hair cells, especially the outer ones, have direct contact with the endolymph only on the upper surface, where the hairs are situated. As shown by us in previous papers this upper surface has a very thick cuticle in which the rootlets of the hairs are found. It

Fig. 1.—Hair cell from guinea pig. The picture is put together (upper and lower half) from two adjacent hair cells. H—two hairs on the cellular surface. The rootlets are not visible. C—cuticle of the hair cell. R M—Reticular membrane. H e—Part of the Hensen body. The lamellar composition described by Engström and Wersäll is not visible. U P—Upper plate of a Deiter cell. V—Vertical surface of the hair cell with the two surface layers. N—Nucleus of the hair cell. D—Deiter's cell. N n—Budformed nervtermination. Enlargement 9000 x. Technic: plastsection according to Sjöstrand.

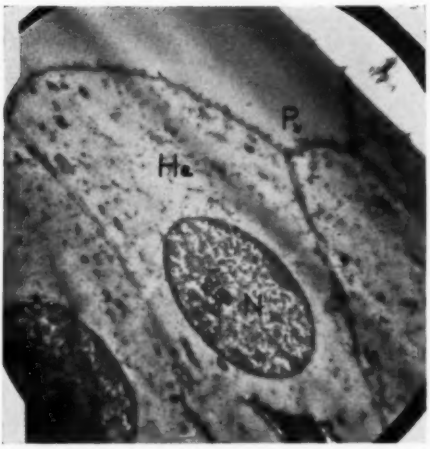

Fig. 2.—Section through some Hensen cells (He) with surface structures (P) and nucleus (N). Enlargement 7000 x.

is not probable that either the hairs or the cuticle has any pronounced resorptive capacity and it is thus not probable that the nutrition of the cells will be intermediated by these structures. The lower end of the hair cells on the other hand is placed in the hollow or cup shaped lower part of the Deiter cells and these cells have a rich mitochondrial and structured cytoplasm well disposed for nutritive functions. These cells have, however, no direct contact with either capillaries or the endolymph. They have on the other hand a direct contact with the fluid in the spaces of the organ of Corti itself (Corti's tunnel, Nuel's space and the outer tunnel as well as the spaces between the hair cells and the slender processes of the Deiter cells). Whatever continuity there is between the endolymph and this fluid is not yet known.

Another possible route for the nutrition can be from the fluid surrounding the hair cells through the long vertical side surfaces of the hair cells.

The important question then arises: How can this fluid be oxygenated and how can nutritive elements reach the hair cells from the endolymph? It is probable that this must be intermediated by

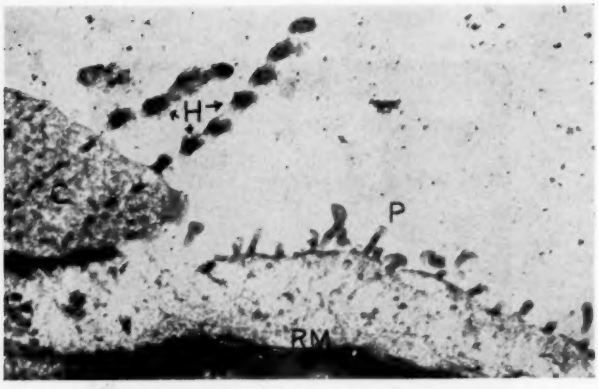

Fig. 3.—Section through the upper surface of a hair cell with an adjacent upper plate of a Deiter cell. Enlargement 28,000 x. C—cuticle P—filiform processes. RM—reticular membrane. H—two rows of hairs.

either Hensen's cells or through the basilar membrane from the spiral vessels. It is quite possible that some part of the nutrition of the hair cells will come the latter way. But how is it with the cells of Hensen? For a better understanding of this question it is necessary to devote some interest to the structure of other resorptive cells.

In the striated layer of the intestines we have a very highly differentiated resorptive organ and as well by light as by electron microscopy it is today established that the striated border consists of a very great number of small finger-like processes with a very thin wall.

In the olfactory region we have another highly resorptive structure. According to the studies of Bloom and Engström, it is now evident that there we have thin walled, finger-like processes in great numbers showing great resemblance to that of the intestinal striated layer.

If a similar structure could be found on the surface of Hensen's cells it would speak in favor of the hypothesis that the Hensen cells are intermediating resorptive elements for the nutrition of the hair cells and the Deiter cells.

In the course of our studies of the structure of the organ of Corti we were much impressed by the constant appearance of a very rich system of thin walled finger-like processes on that surface of

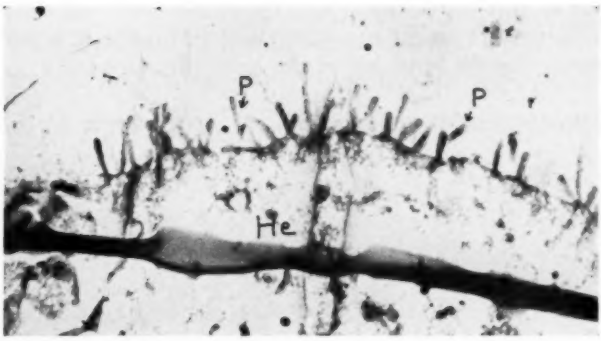

Fig. 4.—A great number of filiform processes (P) on the surface of a Hensen cell. Enlargement 30,000 x.

the Hensen cells which is directed towards the endolymph whereas no such structures are found on the surface directed towards the outer tunnel. The whole "outer" surface of the Hensen cells is uneven, with filiform thin walled processes and these structures are very constant in every guinea-pig cochlea that we have studied. The diameter and length of the filiform processes can be seen in the figures (2-4). They are so small that they are scarcely visible with light microscopy but when we made a very thorough study of a great number of common paraffin and celloidin sections from cochleas we found the "outer" surface of the Hensen cells quite uneven, corresponding well to what we had found by means of electron microscopy.

It was also found that the upper plates or phalanx of the Deiter cells on their free upper surface showed the same type of surface organs. These upper plates, which totally fill out the spaces in the reticular membrane around the hair cells are much larger and have a richer protoplasm than expected from light microscopy.

The appearance of an organ showing similarities to other resorptive organs does not of course give absolute evidence of similar functions. It is quite evident, however, that on the endolymph side of the Hensen cells and on the upper plates of the Deiter cells there is a special differentiation of the cells well adapted to resorptive functions.

SUMMARY

The authors give a description of a rich system of minute filiform processes on the "outer" surface of the Hensen cells and the phalanx of the Deiter cells in the organ of Corti. Their possible functional importance for the nutrition of the hair cells is discussed.

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L

CORROSIVE STRICTURE OF THE ESOPHAGUS: A CASE TREATED BY ESOPHAGEAL RESECTION AND INTRATHORACIC ESOPHAGO-GASTROSTOMY

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Corrosive esophagitis due to lye or caustic soda and the sequelae are relatively common in children; but, by the introduction of radical surgery after the failure of dilatation, these patients now have a greatly improved outlook. This lesion and its sequelae are discussed and such a case is presented here. Treatment was primarily by peroral esophagoscopic dilatations and subsequently when aged three years and three months by esophagectomy.

Any chemical that will cause ulceration of the esophageal mucosa can lead to subsequent stenosis of that organ. The chief offending chemicals are: caustic soda and its commercial equivalents (lye and various cleansing and washing powders), acids, bichloride of mercury, ammonia, and washing soda (sodium carbonate).

Children are more commonly affected than adults.

According to Jackson and Jackson[1] esophageal strictures occur at the levels of anatomical and physiological narrowings in the following order of frequency—at the level of the crossing of the left bronchus, at the crico-pharyngeal sphincter, and at the hiatus. Strictures of the cardiac are rare.

The esophagus is a delicate organ and reacts severely to trauma. Caustic soda and most other corrosives have a great affinity for water and therefore rapidly attack the moist wall causing necrosis and ulceration. The alkalis are even more damaging than acids because of the less efficient means of neutralizing alkalis by body fluids. This difference is also seen with acid and alkali burns of the eye.

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The pathogenesis of corrosive stricture formation of the esophagus has not been clearly described anywhere; but, no doubt, it follows the usual pattern of acute inflammation with ulceration and sloughing, then the formation of fibroblastic tissue which later contracts. The tendency is for the damaged area to be eccentric. This has a bearing on the formation of the stricture and also affects its later dilatability (Jackson and Jackson[1]). A complete concentric stricture from a deep burn is very hard to dilate, as in the case reported.

The stricture, when formed, is paler than the surrounding mucosa and the scar may form a ridge. The esophagus is often dilated above and there may be sacculations or diverticula. There is usually a secondary esophagitis above the stricture from food residues and the region of the stricture itself is subject to periodic attacks of inflammation and edema. In many cases of caustic soda burns the strictures are multiple.

Tucker[2] drew attention to the fact that the esophagus is bowl-shaped above the stricture and funnel-shaped below. This funnel-shape favours his retrograde technique of dilatation.

Those cases in which perforation occurs at the time of the accident are usually quickly fatal.

Unless vigorously treated, complete obstruction of a stricture from a bolus, especially in a child, will be rapidly fatal from dehydration, starvation and acidosis. Treated efficiently, the immediate mortality now is very small. The number of esophageal cripples, however, must still be quite considerable.

Initially there is pain and dysphagia from esophageal reaction. The patient salivates freely and is afraid to swallow. There is often a moderate to severe degree of shock. The lips, gums and mouth are usually burned and desquamated. Gradually the acute stage settles down and he may appear normal for a few weeks. Then increasing dysphagia, regurgitation and vomiting come on with the developing stenosis and, untreated, may go on to aphagia. Loss of weight and dehydration result from the obstruction.

The diagnosis is usually obvious but sometimes the initial burning may be forgotten or ignored and the onset of dysphagia may be delayed even for years. In delayed cases in adults the other causes of increasing dysphagia with regurgitation and progressive loss of weight must be considered. These include cancer, foreign body, pharyngeal diverticulum, and aneurysm with compression. Barium

swallow studies and esophagoscopic examinations will establish the diagnosis.

The objectives of treatment as outlined by Lynch[3] are as follows:

(1) Emergency treatment: To attempt to neutralize the corrosive agent with two per cent acetic acid or vinegar for caustic alkalis and sodium bicarbonate solutions for acids. These are followed by small doses of olive oil or bismuth given at frequent intervals over the first 24 hours. An intragastric tube may be passed at this stage, and, if tolerated, will serve for replacing fluids and for gastric lavage in cases in which the alkali has also burned the stomach. Fluids only are given. If necessary, these are administered parenterally. Further supportive measures for the shock may be necessary.

(2) To maintain a general state of health so that the lesion may heal: This is the continued careful handling of the patient in the early days after the accident. If the patient is unable to swallow after 24 hours, thus indicating a severe esophageal reaction, gastrostomy should be done to permit feeding, at least temporarily, by that route.

(3) To maintain a patent esophageal lumen and to endeavor to restore normal function: Soon after 48 hours from the accident a barium swallow examination should be made to determine the state of the esophagus. Esophagoscopy at this early stage is considered by some to be dangerous; but, if carefully performed and providing the esophagoscope is advanced only as far as the most proximal lesion, it can give useful information as to the degree of damage. Subsequently, the method of Salzer[4] and Bokay[5] (as described by Gellis and Holt[6]) can be commenced. This is the peroral indirect passage of mercury or shot-filled catheters down the esophagus. The patient is persuaded to swallow the well-lubricated bougie and its weight is allowed to carry it down as far as the stomach. It is allowed to remain in place for five minutes. This is repeated daily starting with a small bougie and gradually increasing the size up to the largest possible (about to size 24.F for a young child). It is left down for increasingly longer periods, up to 30 or more minutes. After daily treatment for two weeks, bi-weekly treatments are given for two months and then once monthly for about six months. Thereafter, occasional reviews by barium swallow and esophagoscopy are made.

With this routine the majority of cases seen early heal without any serious stenosis developing. Unfortunately, most cases are not seen by the laryngologist at this early stage and the initiative is lost

by the formation and contracture of fibroblastic tissue. So it is that most cases when seen already have a stricture either long since formed or in the process of developing. They probably have had several attacks of incomplete obstruction which have been overcome but eventually a larger bolus blocks the lumen. If badly obstructed and especially in children, the dehydration and acidosis must receive first attention. Fluids are given, if necessary, parenterally.

Once the emergency is overcome the patient is reviewed by barium swallow or by esophagoscopy. The definitive treatment of the stricture is then undertaken and the following methods have been used:

PERORAL METHODS

(1) Gum bougie. An elastic gum bougie, first stiffened by freezing and then pushed blindly down the esophagus, was originally mentioned by Fletcher in 1831, but it was very dangerous and about it Trousseau, as quoted by Tucker,[2] said: "Sooner or later all cases of stricture of the esophagus die of the bougie."

(2) Weighted bougies. These are the graduated mercury-filled catheters of Hurst. They are well-lubricated and are eased down via the mouth until swallowed. Their passage depends on their weight and they should never be pushed. In suitable cases they are excellent and the patient, even a young child, can be instructed in their use. They are used daily at first, in increasing sizes, but, as the stricture dilates, the intervals can be gradually increased. A review by barium swallow and esophagoscopy is made at regular intervals.

(3) Dilatation under direct vision with an esophagoscope. This is the method advocated by Jackson and Jackson[1] and is commonly used in Australia. The stricture is seen and is dilated with silk-woven flexible-tipped graduated esophageal bougies. Treatments are given every seven to ten days gradually stepping up the size until the largest possible for the age of the patient (24.F-34.F) is passed. Once the stricture remains well dilated Jackson advocates having the patient swallow a suitable sized stomach tube once a week for a considerable period.

(4) String-guided methods. Plummer's method. The patient is persuaded to swallow a silk thread with a lead shot attached. Once the string has passed into the stomach it is used to guide fenestrated olive-tipped bougies of gradually increasing size through the stricture. Once dilated to a sufficient size one of the indirect methods is used to maintain the increased lumen.

(5) Other methods. Forcible divulsion, esophagotomy (internal and external) and electrolysis are only mentioned for completeness and, as far as is known, are not now used.

RETROGRADE METHODS

Ehrlich in 1897 first successfully performed retrograde esophagoscopy via a gastrostomy opening, but forcible dilatation with forceps had been used before this with a high mortality. Billroth and Acker[7] were the first to use retrograde dilators attached to a string. The latter used a rubber tube stretched on a fish-bone support. The tube was led into the stricture and the support removed. The expansion of the rubber resulted in dilatation of the stricture. Acker[7] also first thought to tie the string ends together after treatments. Guisez[8] used stretched rubber tubes for this purpose. Other methods with strings have been used mainly in an endeavor to saw through a tight stricture.

Retrograde bouginage is performed after gastrostomy. The gastrostomy may have been done primarily as an urgent matter soon after the accident or it may have been necessary for increasing obstruction. Again, it may have been made for the direct purpose of retrograde dilatation of a stricture. Whatever the reason, it is an excellent method to approach a stricture. As stated by Tucker,[2] who developed this technique, the stricture is easier to enter from below, it requires less force and is tolerated better by children. It may also result in dilatation of multiple strictures. The difficulty often is to get the string through the stricture. Having the patient swallow a length of silk thread which is first passed through the nose is the commonest method. When it reaches the stomach, which may take several weeks, it is recovered from the gastrostomy fistula. If this fails a lead shot can be tied to the thread which may help it pass. Passing a filiform bougie with the string attached is sometimes used. Jackson and Jackson,[1] Tucker[2] and Hanckel[9] mention various techniques using the esophagoscope retrogradely for patients in whom difficulty is found passing the string. Hanckel has had a urologist, using a cystoscope in the stomach ballooned with water, pass a ureteral catheter through the cardia. The catheter was grasped in the peroral esophagoscope and a string drawn through. Tucker with Chevalier Jackson was able to pass an almost obliterated esophageal atresia using peroral and retrograde esophagoscopes together and forcing a filiform bougie through the area below.

Once the string is passed the ends are tied together and bouginage is commenced. Tucker developed fusiform soft rubber bougies with a loop at each end, ranging from No. 10.F to No. 30.F. One

end is tied to the string and dilatations are carried out once or twice a week. The bougie is permitted to lie in each stricture for from 15-30 minutes. No anesthetic is necessary and the treatments can be carried on at home. After the bougie is withdrawn a new string is left in its place. Once dilatations have begun the patient is able to swallow fluids and semi-solids. The diet should be fortified with added protein and vitamin concentrates. The feeding tube is kept in the gastrostomy merely to maintain its patency. When the dilatations have maintained a fairly large calibre (say about 28.F) per-oral bougies can be substituted and, if they are satisfactory, the gastrostomy can be closed.

RADICAL OPERATION

The development of thoracic surgery, the introduction of antibiotics and the great advances in anesthesia make possible radical corrective procedures for esophageal stenosis. The bougie methods of treatment as outlined above have made dilatation of a stricture very safe and according to Jackson "any patient having any kind of lumen, however small . . . can be cured by dilatation with perfect restoration of function." However, all these treatments take time, often months and years. During that time the patient can carry on some of his usual activities but he must of necessity be restricted. His diet has to be carefully gauged and in the presence of a gastrostomy he is uncomfortable to say the least.

Children, who form the bulk of corrosive strictures victims, have an unhappy outlook. A gastrostomy in these young patients can be very difficult to manage and is not without risks of leaking in the early stages, with subsequent peritonitis. Also, bougie therapy of a stenosed esophagus is frequently attended by attacks of periesophagitis and even mediastinitis. It is not uncommon after a dilatation, especially if there was some attendant bleeding, for the patient to have fever for a few days, and complain of pain. Infection almost certainly enters at the stricture's abraded surface.

Some strictures, especially from a deep concentric burn, can be extraordinarily hard to dilate, and contract very quickly afterwards. This was so in the case to be presented. It is difficult in these patients to maintain adequate nutrition as they are, at best, only able to take fluids and very soft foods. Again, it has been found impossible to pass a string. Permanent gastrostomy is an imperfect solution in these cases, and in young children the prognosis after gastrostomy is not good. As a result the decision may have to be made to remove the atresic segment and to restore continuity by esophagogastrostomy or esophagojejunostomy. When such an operation is being considered,

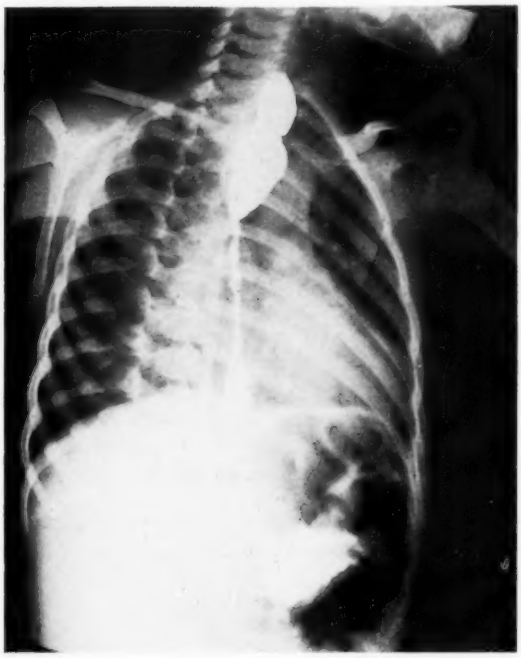

Fig. 1.—Barium swallow 11-2-52 showing the extent of the structure of the esophagus.

gastrostomy should, if at all possible, be avoided otherwise the subsequent operative field will be deranged and mobilization of the stomach will be rendered more difficult and hazardous. If, however, a gastrostomy fistula is already present, it will require closure before undertaking the anastomosis.

A review of the literature on esophageal resection shows that:

(1) In 1894 Bircher advocated an ante-thoracic skin-lined tube to connect the cervical esophagus to a gastrostomy opening.

(2) Torek[10] first resected the thoracic esophagus successfully in 1913. It was for carcinoma. The proximal end was brought out through the neck and a gastrostomy was performed. He connected the two fistulae with a rubber tube.

(3) Yudin[11] described a formidable procedure of what he refers to as the "Roux-Herzen" type of esophagoplasty performed usually in several stages. The esophagus and stomach were by-passed, the jejunem was brought up through a subacutaneous ante-thoracic tunnel and anastomosed end-to-side to the cervical

esophagus. He reported 80 cases of cicatricial stenosis treated this way. In his hands results appear to have been satisfactory from the point of view of low operative mortality and ultimate function.

(4) Other ante-thoracic procedures were described by Longmire and Ravitch,[12] and others.

(5) Intra-thoracic esophagojejunostomy for lesions of the upper third of the esophagus was described by Reinhoff[13] in 1946.

(6) Utilization of the stomach for the anastomosis was first proposed by Fischer[14] in 1926 and first performed by Decker[15] in 1935. Garlock[16] in 1940 reported seven cases of radical operation for carcinoma of the esophagus, and Churchill and Sweet[17] in 1942 reported cases of esophagogastrostomy for carcinoma of the lower quarter of the esophagus.

(7) Sweet[18] in 1946 reported three successful cases of resection of the esophagus and esophagogastrostomy for esophageal stricture.

(8) Cecil[19] in 1949 reported three successful cases of esophagogastrostomy for lye stricture.

(9) Taylor and Clagett[20] in 1949 reported a case of stricture in a child of five years first treated by prolonged dilatation therapy and subsequently by resection and esophagogastrostomy.

The present day methods of treatment can be summarized thus:

For recent corrosive burns, the preventive treatment of stricture, there is a choice between dilatation under direct vision (Jackson and Jackson[1]), and peroral indirect bouginage with weighed bougies (Salzer[4] and Bokay[5]).

For the definitive treatment of a stricture the choice is between dilatation and resection followed by esophagogastric or esophagojejunal anastomosis. Where gastrostomy has not been performed dilatation may be done directly under vision; indirectly with swallowed weighted bougies or by some combination of these. Where gastrostomy has been performed retrograde bouginage (Tucker's method) is used.

REPORT OF A CASE

R. G., was born February 4, 1949.

On December 11, 1951, when aged 2 years and 10 months, he swallowed a mouthful of solution of 2 ozs. of pure sodium hydroxide dissolved in 4 ozs. of water. The solution was to be used in developing photographs and was put to cool in the refrigerator just prior to the accident. He screamed and, within seconds, his father upended a bottle of vinegar in his mouth. He began to cry and vomit. There was no blood in the vomit.

Within an hour he was admitted to hospital where his esophagus and stomach were washed out with weak acetic acid solution. The child was distressed; his lips and gums were inflamed and desquamated but his tongue and palate were not very badly affected. He was tender on epigastric pressure and was salivating freely. After admission to hospital he apparently settled down and took fluids.

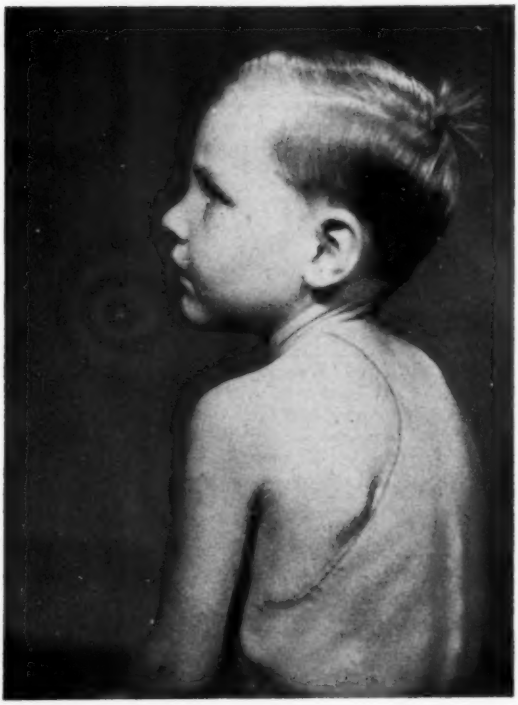

Fig. 2.—Photograph taken Feb. 26, 1953 (nine months after the operation) and showing the situation and extent of the wound and the condition of the patient.

Six days later there was intermittent fever without any obvious cause. The next day there was a melena stool and a benzidine test was positive for blood in the feces. The hemoglobin level at this stage was 10.6 gms per cent.

On January 21, 1952 he was taking fluids and some solids well, but would vomit after swallowing. Two days later he was still vomiting after eating and seemed to cough up the food.

On January 30, he was seen in consultation by one of us and the report was "probably some ulceration and commencing stricture. Thin opaque swallow and esophagoscopy suggested." He was transferred to the Ear, Nose and Throat Service and on the next day the esophagoscope was advanced to a tight stricture at 20 cm from the incisor teeth. Suitably small dilators were procured and on February 7 the stricture was endoscopically dilated with considerable difficulty admitting a No. 14.F Jackson type bougie.

On February 11, the barium swallow showed a well-marked constriction present in the esophagus commencing at the level of the sixth thoracic vertebra and extending downwards for about an inch or so.

Dilatations were carried out at weekly intervals for the first month but at every dilatation the stricture had contracted down to a narrowness that would only admit the No. 10.F dilator. Bleeding always took place even with the slightest stretching and it was difficult to dilate the stricture any further that No. 15.F. However, after each dilatation he was able to eat some semi-solids such as sieved vegetables and fish although his main diet was a fortified high protein milk mixture. He gained weight and no longer vomited. At this early stage his hemaglobin level was between 9.5 and 9.9 gms per cent and the serum chlorides were normal at 610 mgm per 100 mls of serum.

His dilatations were now performed at fortnightly intervals until his discharge to the clinic, but the largest dilator which could be safely passed was No. 18.F. On one occasion a No. 20.F was passed almost through the stricture.

He was discharged on March 20, to attend for dilatations and follow up. At the time he appeared very well and was able to swallow fluids and even some solids which had been first passed through a fine sieve. He was dilated fortnightly for the next two months and then dilators up to No. 22.F could eventually be passed. Even so, on each occasion the proximal end of the stricture was at first found to have contracted down slightly to permit only a No. 10.F or No. 11.F dilator to enter. His mother noticed that his swallowing was impaired toward the end of each interval between treatments. She also stated that he would hold food in his esophagus and at times seemed to have pain getting it down; but, despite this, he appeared to be making progress. He had now been having dilatations for a period of nearly four months during which time he had nine treatments.

However, on May 22 he was brought to the hospital because he had suddenly commenced to regurgitate everything. An esophagoscopic examination was made immediately, but it was found impossible to pass even the finest dilator. The stricture felt hard and inflamed.

An emergency had now arisen as the child was getting no nourishment and was not even able to swallow water. We conferred with the anesthetist and decided that a one-stage esophagectomy was indicated. The alternative would have been a gastrostomy followed by attempts at retrograde dilatation which, if unsuccessful, would have necessitated a second stage esophagectomy.

The decision to perform such a formidable operation on one so young (he was three years and three months of age at this stage) was based on the following:

1. The stricture had proved very stubborn to dilatation. All along it had presented the risk of complete obstruction and this had now occurred.

2. At times during the earlier treatments the patient had elevated temperatures and pain suggestive of periesophagitis and perhaps even mediastinitis. On these occasions he was admitted to hospital, given penicillin, and each time the inflammation settled down.

3. Gastrostomy, at best, would permit feeding, but would not be the permanent answer to his and our problem. It would also increase the risks of a later permanent operation. We felt that if resection and restoration of continuity were performed, this would give him a good chance of normal development.

4. We had Clagett's patient as an example of a similar lesion at the age of five years on whom operation was successfully performed.

5. The child's general condition was still very good but could and would deteriorate rapidly and so would greatly postpone or even prevent the operation.

Two days later operation was carried out.

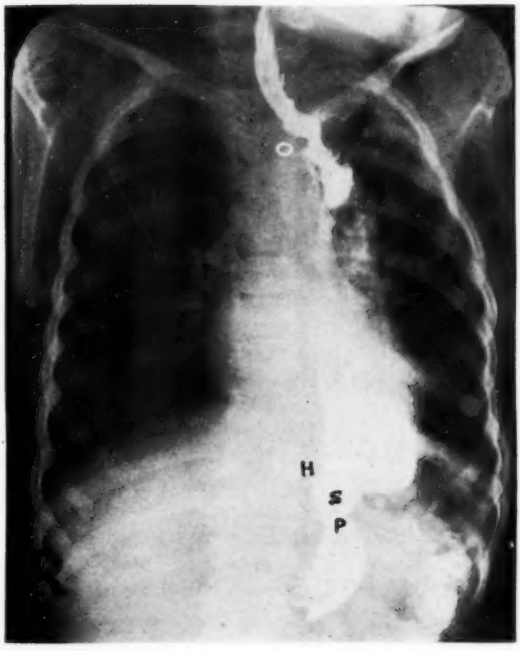

Fig. 3.—Barium swallow Feb. 26, 1953. The site of the anastomosis behind the medial end of the left clavicle is indicated by the indentation on the right side of the barium shadow in this region (0). Some of the barium has already passed through the diaphragmatic hiatus (H) into the abdominal portion of the stomach (S) and even through the pylorus (P).

Operation. With the patient turned on his right side an incision was made medial to and below the left scapula (Fig. 1). About two inches of both the third and seventh ribs were removed and the chest opened. The mediastinal pleura was incised over the lower esophagus which was then followed upwards behind the root of the lung and downwards through the diaphragm to the stomach. The stomach and the lower end of the esophagus appeared normal but that portion of the esophagus behind the root of the lung and arch of the aorta was indurated and constricted.

After opening the left side of the diaphragm the greater and lesser omenta, the left gastric and the left gastro-epiploic vessels,

anastomotic vessels near the cardia, and the short gastric vessels were ligated and divided. The cardioesophageal junction was divided and the cardiac orifice closed with a continuous catgut suture which was reinforced with interrupted sutures of cotton. It was then possible to draw the stomach up to the cupola of the pleura without tension and without affecting its viability.

The esophagus was gradually dissected from its bed to a level just above the arch of the aorta; but, because of adhesions due to periesophagitis, especially behind the arch of the aorta, this was time-consuming. About an inch above the aorta the esophagus appeared normal in diameter, there was no induration of its wall, and there was no evidence of any periesophagitis or mediastinitis. The absence of mucosal changes or a stricture at or above this level was confirmed there and then by esophagoscopy.

The esophagus was divided at this level and the lower portion removed. Continuity was restored by an end-to-side anastomosis of the upper portion of the esophagus to the greater curvature of the stomach about two inches from the oversewn cardiac orifice. Two layers of interrupted mattress sutures of No. 40 cotton were used for the anastomosis. Similar sutures were used to fix the peritoneum of the greater curvature of the stomach to the cupola of the pleura. A catheter was then passed through the nose and down into the stomach.

The opening in the diaphragm was closed about the stomach which was now pulled out and tube-like. After inserting a catheter into the pleural cavity through the lower part of the wound, the opening in the chest wall was closed with catgut for the deeper layers and cotton for the skin. By increasing the pressure of the anesthetic the lung re-expanded. At the same time the end of the catheter was connected to an underwater seal.

During the operation, which occupied four hours, a transfusion of 20 ounces of blood was given although very little blood was lost. On completion of the operation his general condition was quite satisfactory. Thereafter, the blood transfusion was continued very slowly.

The anesthetic, after premedication with atrophine sulphate gr. 1/200, consisted of cyclopropane and oxygen using a Water's cannister and a snugly fitting Magill's intratracheal tube.

The pathologist reported on the excised specimen as follows: "A portion of esophagus, the upper half of which shows a thickened wall and a markedly constricted lumen down to one mm in diameter. The other half appears to be dilated. One piece of the constricted

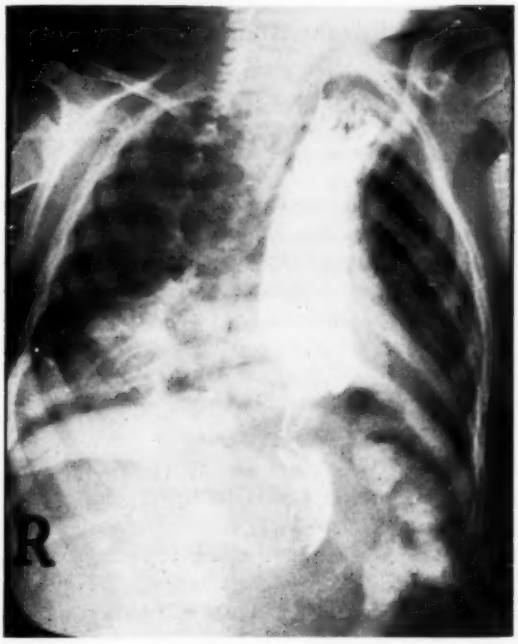

Fig. 4.—Barium swallow Feb. 2, 1953 a few minutes after Fig. 3. This radiograph was taken with the patient in the head down position. The absence of any hold up at the site of the anastomosis or of regurgitation through it will be observed. The height to which the stomach extended is shown.

area in section shows destruction of the mucosa and its replacement by inflammatory scar tissue. Only an occasional fragment of mucosa can be seen, and the inflammatory reaction and fibrosis appears to involve the full thickness of the esophageal wall."

After operation he was given penicillin (50,000 units third hourly), injections of ascorbic acid (100 mgms daily), and morphine sulphate (gr 1/32 when required).

On the evening of the following day the patient's condition rapidly deteriorated when an acute moist condition developed in both lungs. He became cyanosed and lost consciousness within a few minutes. An immediate bronchoscopic examination revealed much thick mucus blocking both main bronchi. This was aspirated

and his condition at once improved and he had no further such trouble. It was probably a reaction to the long operation with the left lung collapsed and to the intratracheal tube.

After another day he again caused some concern as the larynx appeared to be obstructed and there was considerable dyspnea and distress. Tracheostomy was considered but the color remained good and it was not required.

He improved over the next few days. The blood transfusion was stopped on the second day but the penicillin was continued in reduced dosage until his discharge on the forty-first day. Feeding via the nasal catheter was begun immediately and was continued for two days. Small quantities of fluid (one oz. of water every two hours) were then given by mouth. On the fourth day he was put on a milk mixture. From then on the diet was increased and he tolerated food very well. The catheter was removed from the chest of the sixth post-operative day.

The wound healed satisfactorily apart from some superficial necrosis at its lower end which delayed healing for a few days. At first very little could be heard on auscultation of the left side of the chest, and an x-ray examination on June 2 showed that practically the whole of the left hemithorax was opaque, presumably due to a pleural effusion and collapse. A further x-ray two weeks later confirmed this but the child's condition was very good and there was no pyrexia or dyspnea.

A barium meal on June 27 (i.e. five weeks postoperatively) showed that the stomach was occupying a large portion of the left hemithorax and that there was only a little expansion of the lung.

A further x-ray of the chest just before discharge showed a fluid level in the stomach and more re-expansion of the left lung.

He was discharged on July 4 happy and contented. Although he was eating well he then weighed 28 pounds which was 2 pounds, 10 ounces less than before operation. His diet has been a problem since discharge but this is now settling down. He cannot tolerate much milk or fats but apart from that now eats normally. His weight on October 16 was 32 pounds 2 ounces and he looked remarkably well. He had a near accident one night recently when a piece of gristle from a chop caught in his throat but after he went to sleep that passed into the stomach.

An x-ray examination of the chest on October 13, four and one-half months postoperatively, showed the stomach occupying a large part of the left hemithorax but the left lung had, gratifyingly,

reexpanded well, and good breath sounds could be heard over most of the left side of the chest.

On February 26 a barium swallow examination was carried out and it was reported that "the stomach is mainly a thoracic organ with its fundus and cardioesophageal junction at the level of the third thoracic vertebra. The esophagus was examined by barium bolus and barium meal. Both passed freely down the esophagus and through, into the stomach, without any stasis whatever. Occasionally on screening there was a small ledge of barium at the level of the cardioesophageal orifice, probably due to a small amount of barium being trapped in a ridge of tissue but it is very small and does not interfere in any way with the downward passage of either the bolus or the fluid. The pylorus is intra-abdominal, the prepyloric region of the stomach passing through the esophageal orifice of the diaphragm. There is a slight constant constriction, as would be expected, of the stomach at this level and there is a little stasis of the meal at the pylorus; but, as there was no fluid residue in the fasting stomach and no dilatation of the stomach, the degree of stasis is not marked." (Figs. 2 and 3).

During the operation the stomach, rather than the jejunum, was used for the anastomosis as it came up to the top of the chest with less tension. Such an esophagogastric anastomosis always carries the risk of regurgitation and esophagitis; but, fortunately, there has yet been no indication of this happening. At the time of submitting this paper for publication (ten months after the operation) his general condition was excellent and he was eating very well.

SUMMARY

Corrosive esophagitis and its sequelae are discussed.

A case of stricture of the esophagus due to swallowing caustic soda in a child of two years and ten months of age is presented. Five months later after repeated attempts at dilatation he was treated successfully by resection of the lower two-thirds of the esophagus, continuity being restored by anastomosis of the stomach to the upper end of the esophagus above the arch of the aorta. In the ten months since, his progress has been eminently satisfactory.

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STUDIES OF DISABILITY OF THE PHARYNX RESULTANT FROM POLIOMYELITIS

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The concern of physicians caring for patients ill with bulbar-pharyngeal polio has been directed toward methods of effectively replacing the function of swallowing by postural drainage and aspiration, or toward circumvention of the pharynx as a portal of respiration by the procedure of tracheotomy.[1-4] The problem of early diagnosis of this involvement in polio has persisted, and the disability of swallowing still occasionally remains undiscovered until the patient experiences an episode of acute pharyngeal obstruction, severely complicating the further course of this disease by reason of transient hypoxia, atelectasis, and the patient's remembered alarm. A further problem of medical concern is that of the increasing number of patients who continue to have residual disability of swallow or voice which persists prolongedly after the maximum of recovery has been achieved. In a preliminary assay of persistence of sequellae of bulbar poliomyelitis earlier,[5] an unexpectedly high incidence of persistent or recurrent disability of the pharynx is noted. It was found that 28 (59.6 per cent) of 47 such patients had continued or recurrent disability 17 or more months after their acute illness.

These studies are an amplification of the previous report and are concerned with the observation of the polio-paresed pharynx by visual inspection and by cinematography, and the correlation of these observations with disability of pharyngeal function. Two general and inconstantly combined patterns of partial paralysis and associated disability of function have been distinguished. The more common is that of weakness of the levator vili palatini muscle, with associated impairment of the palato-pharyngeal sphincter. This results in a "nasal" quality of speech and possibly in nasal regurgitation of swallowed fluids. The less common but more critical paralysis is that of the pharyngeal constrictor musculature, associated with disability of swallow.

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These observations of the polio-paresed pharynx are related to a study of the anatomy and motor function of the upper pharynx by cadaver dissection and by cinematographic study of patients after maxillo-facial surgery, reported in a preceding publication.[6] The further contribution of these studies of the polio-paresed pharynx to understanding of the mode of action of this organ are discussed.

These observations are applied to the diagnosis and general estimation of severity of bulbar-pharyngeal poliomyelitis. The problems of patients having persistent disability of the pharynx as sequellae of poliomyelitis are reviewed, and routines of their special medical care are suggested.

PATIENT OBSERVATIONS

A total of 28 patients were selected for this study of polio-paresis of the pharynx. Each had survived acute bulbar pharyngeal poliomyelitis between six months and five years before these observations, with the exception of one woman 31 years of age who had severe acute bulbar poliomyelitis following tonsillectomy at the age of five years. At the time of this study the patients ranged in age from 6 to 31 years. The validity of the original diagnosis of acute poliomyelitis was confirmed by review of hospital records in all but the latter patient. These patients continued to have evidences of disability of the pharynx as sequellae of this disease. Additional criteria for selection of these patients was their own willingness to return for repeated examinations, and their cooperation in the tedious procedures of visual and cinematographic examination of the pharynx. Twenty-two of these patients were seen in a special out-patient clinic and six were seen in the hospital in which they were continuing their convalescence.*

General medical history and the history of the episode of acute poliomyelitis and polio-disability were recorded routinely on standard forms prepared for this purpose. Examination was directed principally to the area of the mouth and pharynx and, as an extension of the observations in the preliminary series previously reported,[5] the motions of the posterior and lateral walls of the pharynx were studied during motions of phonation. Cinematographic studies of the pharynx were obtained in 16 subjects, using the camera, illum-

*This interest was continued from 1947 to 1949 at the University of Minnesota Hospitals, the Minneapolis General Hospital and the Elizabeth Kenny Institute, Minneapolis, and subsequently at the Salt Lake General Hospital, Salt Lake City.

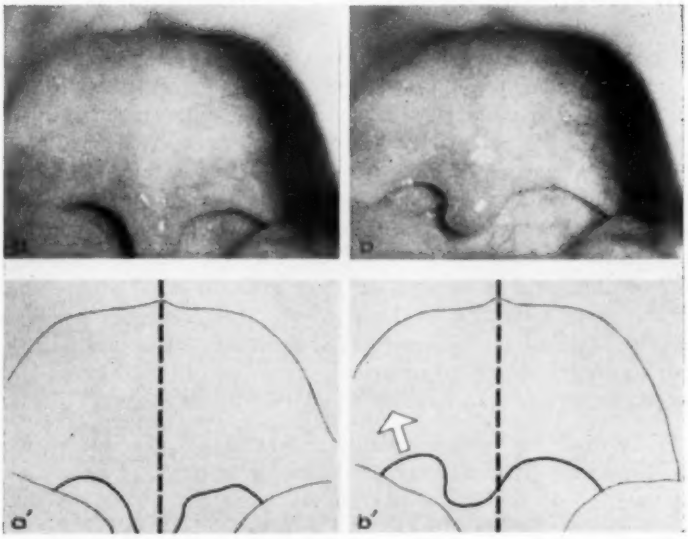

Fig. 1.—Demonstration of weakness of levator palatini on patient's left (right side in illustration) by selective elevation of the left side of palate and leftward abduction of mass of palate. The deviation of the uvula toward the patient's right evidences a coincident weakness of the intrinsic muscles of the palate and the uvula on the patient's left.

ination and patient-positioning techniques employed in the study of the surgically exposed pharynx, as previously described.*[6]

Disability of the function of the pharynx in these patients was variably combined with polio-paresis in other areas of cranial innervation, particularly in the face, with impairment of muscles of mastication and of the tongue noted in diminishing incidence. Impairment of function of the larynx was the most common coincident disability. The incidence and severity of this disability was not evaluated in this study.

Abnormalities of motion of the palate were seen in 22 of these subjects. The most common and most obvious form of this abnormality was that of asymmetrical action of the levator vili palatini muscle, causing an asymmetry of elevation of the palate during

*Studies of action of the normal and polio-paresed pharynx by means of pressure recording techniques and of serial roentgenograms are in progress and will be reported at a later date.

phonation and possibly causing the palate to peak sharply on the side of the stronger levator (Fig. 1). Asymmetrical weakness of the levator was thus identified in 20 patients and bilateral levator weakness, demonstrated by a persistent pendant position of the palate during phonation, was found in two subjects. Paresis of the tensor muscles of the palate was not identified in these patients. Evaluation of action of the palato-pharyngeus muscle was made difficult by scarring of the palato-pharyngeal fold which was found commonly in the 20 of the 28 patients of this study who had had tonsillectomy months or years before their episode of acute bulbar poliomyelitis. Consequently, weakness of the palato-pharyngeus muscle was separately distinguished in only six of these patients, in whom it was coincident with ipsilateral weakness of the levator of the palate and the constrictor of the pharynx. The palato-glossus muscles in their folds showed little or no independent motion in the circumstances of this study, and no disability was identified in them.

The disability of the patients in whom the principal weakness was that of the levator of the palate, was that resulting from abnormal patency of the palato-pharyngeal sphincter. This has shown most commonly as a nasal quality of speech. This disability was present consistently in five patients and intermittently in 14 others at the time of examination. In some having a fairly severe degree of unilateral weakness of the levator muscle there was no nasality of speech. Nasal regurgitation of fluids during swallow was found in general correlation with nasal speech, but was less common, being reported as an intermittent disability in 14 subjects.

Weakness of the pharyngeal constrictor muscle was found in 23 of these patients. This weakness was asymmetrical in 18, in whom it was manifested by an abnormal sideward motion of the posterior pharyngeal wall toward the less involved side with effort of phonation, and a return motion toward the more involved side with termination of effort (Figs. 2, 3). This transverse motion of the posterior pharyngeal wall was seen only with stronger efforts of phonation of tones of medium or high pitch. By reason of the brevity of the motion and the particular circumstance of its occurrence, this sign had not been noted in these patients before cinematographic studies were obtained. Subsequently, this sign was readily distinguished in direct visual examination in these and in many other poliomyelitis patients having like patterns of disability. In association with this lateralward motion of the posterior pharyngeal wall it was found that, whereas the lateral pharyngeal recess of the more normal side fills in the usual manner, with the appearance of a varied pattern of corrugation in the more lateral portion of the wall, the lateral

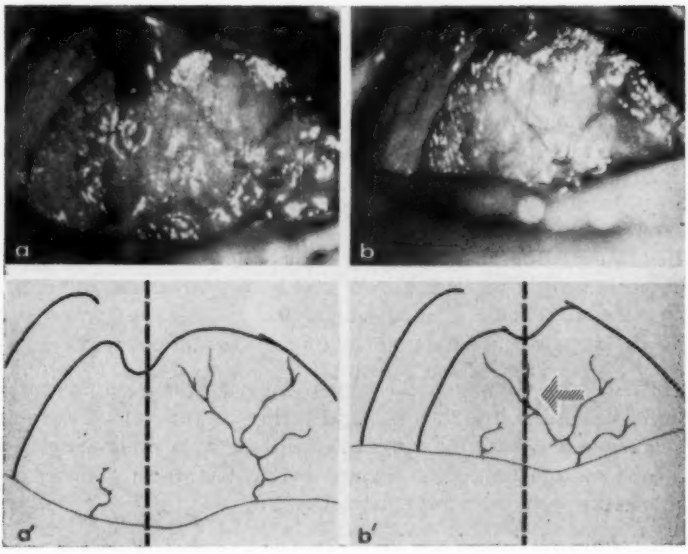

Fig. 2.—Demonstration of Transverse Shift of Posterior Pharyngeal Wall Incident to Phonation. Cinema frame "a" and diagram "a'" illustrate structures of pharynx at rest. Note distortion of palate by post-tonsillectomy scar. Cinema frame "b" and diagram "b'" illustrate positions during high pitch phonation. Note adduction of palatopharyngeal fold and filling in of lateral pharynx on patient's right. There is little motion of the palatopharyngeal fold and no filling of lateral pharynx on patient's left. Transverse shift of the posterior wall is shown by relative position of vessels in two positions.

recess on the paresed side did not fill in but was diminished in size by a pulling of the uncorrugated posterior wall toward the opposite side. This differential filling of the lateral recess of the pharynx on the unparesed or less paresed side has seemed at times to be more prominent than in the normal pharynx.

Weakness of the constrictor musculature of the pharynx was found to be associated with an exaggeration of the evidences of abnormal patency of the palato-pharyngeal sphincter in those patients in whom there was also weakness of the levator muscles, and with a difficulty in swallowing, with or without nasal regurgitation. The difficulty in swallowing was reported as occasional and in mild degree in ten patients frequent and moderate degree in six patients and con-

stant in five patients. In the latter five patients, swallowing was performed slowly, cautiously and with an increased awareness of the act. The patients having dysphagia of moderate or severe degree developed habits of chewing their food thoroughly, and commonly "washing it down" with liberal quantities of fluids. In spite of these precautions, they were liable to recurrent episodes of choking incident to the eating of solid foods. These episodes tended to occur particularly in times of illness, fatigue and emotional distress. The patients having dysphagia also became peculiarly selective in their choice of diet, avoiding strongly flavored foods and liquids, and also often avoiding milk. They would report that the former "burned" persistently in the pharynx, and that milk caused an increase in pharyngeal secretions. The flavor chocolate was also reported to cause an excessive accumulation of mucous in the pharynx.

Those with asymmetrical constrictor weakness commonly developed a characteristic turning and tilting of the head toward the more normal side of the pharynx in association with swallowing. This associated motion tended to become habitual in these patients, and some of them lost their awareness of it.

A further problem of these patients was that of the accumulation of secretion in the pharynx during sleep. Their family associates would report snoring and a "gurgling" quality of respirations during sleep. The patients reported occasional episodes of wakening in alarm, to find themselves sitting upright and coughing strongly. These episodes resembled those of paroxysmal nocturnal dyspnea, as found in patients in cardiac failure. This difficulty was present particularly at the time of respiratory infections. In those having a unilateral disability of the pharynx, the problem of "pooling" of secretions in the pharynx could be diminished by having the individual sleep on the side opposite that of the greater pharyngeal weakness.

In the follow-up visits, a method of evaluation of the strength of swallowing was devised, which consisted of application of downward traction upon the related mass of structures attached to the hyoid bone, including the larynx, base of tongue and attached walls of the pharynx, by means of the forefinger and thumb inserted in the notch between hyoid bone and cricoid cartilage. In normal subjects, the strength of elevation of this "hyoid mass" was sufficient to pull the larynx upward from between the clasping fingers. Such was also true of those poliomyelitis patients who had no impairment in swallowing in the course of their disease. However, those who had such disability demonstrated a deficiency of this motion, and it was possible in these subjects to hold the larynx downward in its

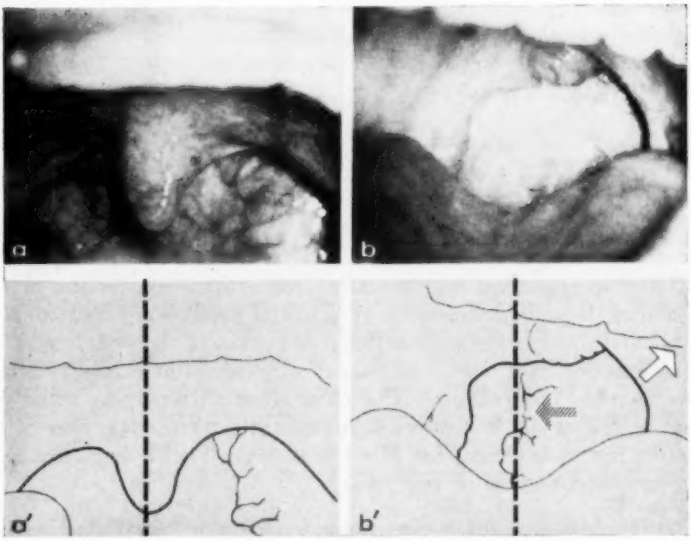

Fig. 3.—Demonstration of Transverse Shift of Posterior Pharyngeal Wall and Filling of Lateral Pharyngeal Recess Incident to Effortful High Phonation. Cinema frame "a" and drawing "a'" illustrate pharynx at rest. Cinema frame "b" and drawing "b'" illustrate positions during phonation. Note palate is elevated and drawn toward patient's left—in this instance an effect of scarring in area of left palatopharyngeal fold. The patient's right palatopharyngeal fold moves medialward and the right lateral pharyngeal recess fills. The posterior pharyngeal wall moves toward the patient's right. This patient has weakness of the left pharyngeal constrictor. Evaluation of the muscles of the palate is obscured by scarring as sequel of tonsillectomy.

position of rest or to restrict its elevation to a minimum. With this failure of elevation, there was also an exaggerated difficulty in swallowing, with nasal regurgitation of the test sample of water or deficiency of descent of the swallowed mass, the latter resulting in a partial spilling into the larynx. This test was also useful in the evaluation of the degree of recovery of the function of the pharynx in general. Thus, in those subjects in whom nasal regurgitation of liquids had become infrequent, the disability could be demonstrated again by this method, indicating that recovery of the function was still incomplete.

As might be expected, the most severe disability of the pharynx was found in those five patients having severe bilateral weakness of

the pharyngeal constrictor muscles. The disability was greater than in those having lesser degrees of weakness. Three of the patients who had severe weakness of both the constrictor and levator muscles had a distinctive pharyngitis sicca.

DISCUSSION

The evaluation of neurologic impairment of the pharynx is peculiarity difficult by reason of the complexity of its anatomical arrangement, the complexity of its innervation and the rapidity of its principal function, that of swallowing. In the clinical observations here described, disability of the musculature of the pharynx incident to phonation has been identified and the significance of this disability becomes evident in speech and swallowing and in such incidental signs as the accumulation of mucus in the pharynx or the dessication of its mucosa. A variety of patterns of disability of the pharynx has been found. The most common weakness noted was that of the palate, but the most important weakness was that of the pharyngeal constrictor, for this was associated with impairment of the critical function of swallowing.

The lower motor neurons innervating the intrinsic muscles of the pharynx: the levator, pharyngeal constrictor, palatopharyngeal and salpingopharyngeal muscles are found in the nucleus ambiguus, and are distributed to these muscles via the glossopharyngeal and vagus nerves.[7] The extrinsic musculature suspending the pharynx and the closely related mass of the hyoid, the larynx and the base of the tongue is innervated by this nucleus ambiguus, via the glossopharyngeal nerve (supplying the stylopharyngeus muscle), by the trigeminal nucleus and nerve (supplying the mylohyoid, geniohyoid, and anterior belly of the digastric muscle) and by the facial nucleus and nerve (supplying the stylohyoid muscle and the posterior belly of the digastric muscle).[7] Swallowing is initiated by the action of the tongue, supplied by the hypoglossal nucleus and nerve.[8, 9] The contribution of the tensor of the palate, supplied by the trigeminal nerve, to the function of the pharynx is not yet clear.

The impairment of the pharynx found in these patients surviving acute poliomyelitis was that resulting from lower motor neuron involvement. Thus, the impairment was characterized by simple paresis or paralysis, rather than by dyscoordination. Though abnormal movements were distinguished they were not the result of imperfect organization of motor activity in time or in muscle distribution. Rather, the abnormal movements which were observed were apparently effective in compensation for the paresis or paralysis with which they were associated. Thus, in the patients having asym-

metrical weakness of the constrictor, the shift of the posterior pharyngeal wall toward the more normal side of the pharynx diminished the lateral recess on the lax side of the pharynx. There was no noted evidence of contracture in the intrinsic muscles of the pharynx except that associated with the trauma of tonsillectomy.

If we conceive of the pathologic process which causes the paralysis of poliomyelitis as one of discreet or focal necrosis irregularly distributed in the nuclear areas,[11] then the variety of patterns of paralysis of intrinsic muscles of the pharynx found in this study is that which might be expected to result from involvement of the nucleus ambiguus in this disease for, though the motoneurons of this nucleus are somewhat concentrated in its proximal bulb, the remainder are distributed in its elongate portion. The particular pattern of representation within the nucleus ambiguus has not been determined. However, the inconstant association of paralysis of the levator and the constrictor muscles implies that their motoneurons are remote. Upon the basis of patterns of pharyngeal disability and of the site of necrosis within the nucleus ambiguus Baker, Matake and Breen[11] suggested that the rostal "bulb" of the nucleus was concerned with the function of the palate, and the more distal "tail" with the function of the constrictor muscles of the oro- and laryngopharynx.

It is apparent that this "lower motor neuron" type of disability is not peculiar to acute bulbar poliomyelitis and its sequellae. Any lesion causing impairment in the area of the nucleus ambiguus or its efferent distribution may occasion like disability of the pharyngeal musculature which it innervates. Thus, similar patterns of disability may result from post-diphtheritic paralysis or botulinus intoxication. However, the development of this type of pharyngeal disability in association with the clinical syndrome of acute poliomyelitis makes this diagnosis highly probable. Physicians, alert to the evidences of paralysis in the pharynx such as those here noted, will at times be able to identify this critical involvement in the disease poliomyelitis before any impairment in the function of swallowing has appeared, and, by institution of appropriate measures of therapy, may prevent the exaggeration of difficulty which results in these patients from successive episodes of respiratory obstruction.

DISABILITY OF PALATO-PHARYNGEAL CLOSURE

The action of the pharynx in speech differs considerably from that in swallowing. In the modulation of speech, the upper pharynx is in continual partial motion, with the palate poised in mid-position or varying slightly from it, and the superior pharyngeal constrictor

muscle varying its partial closure of the palatopharyngeal sphincter.[12] These fine modulations of voluntary function are directed from the cerebral cortex. In contrast, the act of swallowing is abrupt, forceful and more nearly uniform in successive performances. The whole pharynx and its suspensory musculature particiate in a brief but powerful motion of elevation. The act of swallowing may be intentional, but usually occurs involuntarily as a reflex initiated by stimulation of the pharyngeal wall.[13] Though a center initiating swallowing has been identified in the anterior central gyrus, the rapid succession of actions by which it is accomplished is probably integrated in the brain stem.[14]

The difference in the manner of performance of the pharynx in speech and in swallowing is well shown in the circumstance of partial paralysis in the intrinsic muscles of the upper pharynx. Thus, in those patients having unilateral weakness of the levator muscle and normal function or slight impairment of the superior constrictor, there was commonly a nasal quality of the voice, indicative of an abnormally patent palatopharyngeal valve. However, in many of the patients having this slight degree of involvement, there was no nasal regurgitation of swallowed liquids. This disparity between disability in speech and in swallowing in patients having a weakness of the palate has been noted previously by Freijstadtl[15] and by Maher.[17] This situation is seemingly incongruous, for the intra-pharyngeal pressure developing incident to swallowing has been determined by Falk as 20 cm of water,[9] a value many times that which occurs in speech. The probable explanation of this variation in disability is that in common circumstances of speech the pharynx functions but partially, whereas in each swallow the pharynx functions with greater range and force of motion and with reinforcement of its levator action by contraction of the extra-pharyngeal muscles. With this greater effort, the palatopharyngeal valve is able to close effectively despite the greater intra-pharyngeal pressure. It is probable that the superior pharyngeal constrictor muscle makes a considerable contribution to this closure incident to swallowing.

The general significance of the superior pharyngeal constrictor in palatopharyngeal closure is shown by the greater disability of this closure in the circumstance of combined constrictor and levator muscle weakness, as compared to that of levator weakness alone. In those patients having both of these muscles paresed, there is an accentuation of the nasal quality of speech and more common nasal regurgitation of liquids.

In the circumstance of deficient elevation and posterior displacement of the palate incident to levator muscle weakness new im-

portance is given to the ridge of Passavant, the shelf-like anteriorward projection of the posterior pharyngeal wall and the corresponding medialward projection at the level of palatopharyngeal closure. In cinematographic of subjects having exposure of the pharyngeal wall by anomaly or surgery,[6] marked differences were found in the prominence of Passavant's ridge as formed incident to speech. The differences may reflect actual differences in arrangement and quantity of the superior pharyngeal constrictor muscle, which contributes to the palatopharyngeal valve or sphincter. These individual differences may partially account for variations in degree of disability of the upper pharynx among patients having similar degrees of paresis.

Additional causes of variation in degree of disability include such extraneous or imposed circumstances as fatigue, psychic distress or intercurrent illness. The significance of these added factors upon the disability of the pharynx was found to be considerable, often evoking spontaneous comment from the subjects or other informants in this study. The accentuation of disability was equivalent to a reversion to an earlier stage of recovery.

The accentuation of the degree of nasality of speech incident to acute upper respiratory infections is particularly noteworthy, for the direction of disability is opposite to that which occurs in normal subjects, who develop an exaggerated palatopharyngeal closure during such illnesses, so that "explosive" sounds, resulting from such closure, become excessively prominent.[18]

DISABILITY OF THE ORO- AND LARYNGOPHARYNX

By reason of its conspicuousness, the clinician is liable to overemphasize the weakness of the levator muscle of the palate in bulbar poliomyelitis. This paralysis of the constrictor musculature of the pharynx is more critical than that of the levator of the palate, as the function of swallowing is thereby impaired. The pharynx is the common portal of deglutition and respiration, and these two entering streams must be strictly segregated. The alternation of swallowing and respiration is accomplished by a specific reflex mechanism, whereby the stimulus of food in contact with the lateral pharyngeal wall inhibits the next succeeding inspiration.[8, 9] This alternation depends, however, upon adequate emptying by the pharynx of the liquid or semi-liquid bolus impelled into it from the mouth. The pharynx must also empty itself periodically of the mucoid mass accumulating within it in intervals between feeding, as an effect of continuous ciliary propulsion of sheets of mucoid secretion in the trachea, larynx, paranasal sinuses and nasal cavities.[19]

The act of swallowing differs importantly from that of speech in that in the former there is a minimum of function which is essential for the success of the act. This minimum may be defined as a complete emptying by the pharynx of its current bolus of food, so that succeeding respirations are unobstructed. The pharyngeal function of many who have survived bulbar poliomyelitis is marginal in this respect. The disabilities which occur in patients having weakness of the constrictor muscles and resultant impairment of swallowing are those which are to be expected. The greatest difficulty is in eating, when the subject is liable to swallow incompletely and to aspirate the residual pharyngeal contents into the larynx with the next breath, causing choking.

As observed in this study, the patients having difficulty in swallowing consistently had asymmetrical or bilateral weakness of the pharyngeal constrictor muscles. In those having unilateral constrictor weakness, this was identified by an abrupt transverse motion of the posterior pharyngeal wall toward the more normal side with onset of moderate or strong effort of speech. A similar abnormal motion of the posterior pharyngeal wall had been described previously in the circumstance of pharyngeal paralysis by Bönninghaus.[20] There was also a persistence of the lateral recess on the affected side. In those having bilateral severe constrictor paralysis, the transverse shift of the posterior pharyngeal wall noted above was absent, and identification of the paralysis was made upon observation of the lack of normal elevation of the posterior and the lateral pharyngeal walls, with persistence of the lateral pharyngeal recess of each side.

The mechanism of swallowing has not yet been fully determined, and its study is rendered difficult both by the rapidity of the act and the variation in the mechanisms in different species.[10] The clinical observations here reported indicate that the constrictor musculature of the pharynx participates actively in the conveyance of food to the esophagus. This is shown by the above-noted coincidence of disability and constrictor paralysis and, more particularly, by the persistence of food selectively on the side of the pharynx which is paretic.

The disability resulting from the weakness of the pharyngeal constrictor musculature is probably exaggerated by weakness found in the associated palatopharyngeus and salpingopharyngeus muscles, as well. The precise contribution of each of these muscles to the function of the pharynx is not yet understood. The palatopharyngeus must act as an adductor and elevator of the lateral pharyngeal wall and, if inferences made in previous cinematographic studies of

the motion of the hemipalate and palatopharyngeal folds in adult cleft-palate subjects are correct, the palatopharyngeal folds in the normal subject may join in the midline to form an additional barrier between oro- and nasopharynx.[6] Negus,[10] mentions the palatopharyngeus muscle as participating in the closure of the palatopharyngeal isthmus. This statement, applied to the human, is apparently derived from homologic comparisons.

Correspondingly, as the salpingopharyngeus muscle follows the inferior border of the posterior wing of the tubal cartilage upward, medialward and posteriorward incident to swallowing and speech, it acts as a levator of the portion of the pharyngeal wall into which it inserts and also contributes to the medialward motion of the lateral pharyngeal wall at the level of the palatopharyngeal closure and in the oropharynx. Both the palatopharyngeus and the salpingopharyngeus muscles are integral elements of the complex and overlapping musculature of the pharynx. Isolated impairment of a single muscle of either of this pair is probably of little significance in total function of the pharynx, but such impairment is certainly of additive effect in a general paralysis of this organ.

The contribution of the extrinsic pharyngeal muscles to the elevation of the pharynx is apparently of increased importance in the circumstance of paralysis of the intrinsic musculature of the pharynx. As noted in earlier studies of the pharynx exposed by surgery or anomaly, elevation of the pharynx occurs in variable degree incident to its modulation of speech, and strong abrupt elevation of the pharynx occurs in swallowing.[6] The adequate elevation of the pharynx and of the "hyoid mass" attached to it has been found in partially paralyzed poliomyelitis patients to be essential to swallowing. Thus, if slight downward traction was applied to the larynx, the elevation of the hyoid mass was found in some patients to be of less than normal strength, and the attempted swallowing was impaired. The pattern of the impairment so demonstrated was that which the subject had experienced at an earlier stage of recovery. Thus, if he had previously experienced nasal regurgitation, the disability appeared again in this test of swallowing. If the subject had previously had an incomplete swallow, with persistence of food in the pharynx, this likewise recurred. Slight disability in either pattern became exaggerated by the procedure of this test. The importance of the contribution of these extrinsic elevator muscles to the act of swallowing has been pointed out by Negus.[10] In an analogous study of patterns of post-diphtheritic paralysis of the area of the pharynx, Maher[17] also found weakness of these "suprahyoid" muscles to be associated with disability in swallowing.

The associated motions of tipping and turning of the head which developed in these subjects incident to swallowing were of particular interest. These motions were first found at the time of difficult and effortful swallowing. They were clearly non-imitative, yet appeared nearly identical in different subjects having like patterns of asymmetrical weakness of the constrictor muscles. The direction of tipping and turning of the head was consistently opposite to that of the side of the weaker constrictor. These motions apparently facilitated the act of swallowing, at least in the earlier stages of disability. The manner in which this facilitation occurs is not clear, but it probably results from one or more of the following mechanisms:

1. This head motion is perhaps effective in diminishing the lax lateral pharyngeal recess by elongation of that side of the pharyngeal tube, and thus diminishing the area in which food may accumulate and from which it can be readily extruded.

2. This head motion may well place the extra-pharyngeal levator muscles which are unparesed in a more advantageous position for their effective action. Thus, they may be better able to accomplish the elevation of the total "hyoid mass," which elevation is so essential in swallowing.

3. An additional mechanism which is probably in action in this adaptive movement is that of the direction of the bolus of food into the less impaired side of the pharynx. In the normal pharynx, the bolus in transit is split at the epiglottis and descends through the lateral hypopharynx in parallel streams, which converge again at the constricted area of the pharyngeal-esophageal junction.[9] It is to be expected that turning the head to one side will cause a deviation of the epiglottis within the pharynx toward the opposite side, resulting in the deflection of a greater proportion of the bolus to the same side as that toward which the face is turned. Thus, in the subjects having disability of one side of the pharynx, their adaptive turning and tipping of the head toward the opposite side would result in a deflection of food away from the weaker and into the stronger side.

In careful observation of subjects having post-diphtheritic paralysis of the pharynx Maher[17] found that those having a pattern of unilateral weakness of the levator of the palate, and resultant patency of the palatopharyngeal sphincter, acquired motions of the head similar to those noted in the subjects herein described, which apparently diminished their disability of speech. This particular improvement of a disability of speech was not noted in the subjects of this study.

Among the incidental problems in severe paralysis of the pharynx is pharyngitis sicca. This results apparently from abnormal inspiratory air currents. As pointed out by Proetz,[19] the soft palate in its usual mid-position acts as a baffle which disseminates the inspired air in a series of eddies through the pharynx and adjacent parts of the mouth. The air is thus exposed to a relatively large mucosal area, as a result of which it is warmed and humidified. It is reasonable to suggest that in the circumstance of a widely open palatopharyngeal isthmus, resulting from severe paralysis of the levator and superior constrictor muscles, the inspiratory stream impinges disproportionately upon the posterior and lateral walls of the oropharynx, causing excessive drying of the mucosal surface in this area. This is comparable to extensive excision of the palate, resulting likewise in pharyngitis sicca in a like location.[21]

GENERAL THERAPEUTIC CONSIDERATIONS

By reason of the increasing numbers of bulbar poliomyelitis patients with pharyngeal disability who are urgently in need of medical assistance, much experience has been accumulated and recorded regarding the care of these patients during their acute illness.[1-4] There has been less attention directed toward the medical measures employed in the care of these patients in the weeks and months of their convalescence, though such care may contribute much to their early rehabilitation. In addition to the general medical and physiotherapeutic routines of care appropriate to any patient convalescent from acute poliomyelitis, these individuals having residual pharyngeal disability deserve the following particular considerations:

1. *Guided experience in eating and swallowing.* The resumption of oral feeding is a time of much concern on the part of both the patient and the physician. The patient is usually uncertain about the recovery of his ability to swallow and both the patient and the physician are apprehensive about the threat of repeated episodes of choking and hypoxia. The schedule and graduation of feedings is individualized for each patient. A satisfactory succession of early feedings may include:

   a. Water and isotonic saline solution,
   b. Uncarbonated beverages and unsweetened fruit juices,
   c. Bouillon and thin gruel.

The subsequent graduations of diet through the range of soups and gelatin preparations, mashed and pureed foods, may be joint selections on the part of physician and patient. The particular later choices should be determined upon the basis of the physical character

of the food. Crisp meats, pastries, fruits and vegetables are added only after the act of swallowing becomes nearly normal, and such foods may be permanently omitted from the diet of those patients who have a persistent disability in swallowing. Strongly flavored foods also should be introduced later.

In the face of a patient's marginal abilities to swallow, the physician is tempted to temporize and to delay gavage feeding. This may result in malnutrition and a further accentuation of the disability. It is advisable to estimate routinely the nutritional requirements and, as soon as the acute febrile illness with its anorexia and nausea is past to insure the nutritional requirements by gavage feeding as needed. The adequacy of the diet should be confirmed by weighing the patient at weekly intervals, and given prime consideration. Adequate diet brings about improvement in morale, increased recovery of function and increased resistance to fatigue.

2. *Adequate psychological support.* The disability in swallowing which these patients have experienced has been the occasion of alarm rendered more acute by the hypoxia resulting from respiratory obstruction. This has perhaps been made even more alarming by tracheotomy and artificial repiration. Throughout this period of alarm, the patient has often been deprived of personal communication by reason of disability of speech. Furthermore these patients frequently recapitulate their anxious moments, in later episodes of choking on food during meals, or on awakening at night with a mucous-filled pharynx.

It is apparent that the attitude which these patients will subsequently develop toward their disability is established during the acute stage of their illness and in the first weeks of their convalescence. Consistent care and guidance of the patient in this period by physicians and nurses familiar with this problem has proven to be of inestimable help to the patient. Attention must be directed to the prevention of hypoxia by aspiration of the pharynx when indicated, and by adept teaching of patience and caution during the early stages of drinking and eating. Group therapy has proven particularly useful in this situation and, when feasible, patients of like disability should be placed in common hospital wards.[22]

Attention to the attitude of the patient toward his persistent disability must be an important part of medical follow-up after discharge from the hospital. The occasion of the return of these patients to their home environment may be a time of discovery of the extent of their disability of speech and of eating, so that they may assume solitary habits, particularly at mealtime.

The nasality of speech which is found persistently in some of these patients is at times a social problem, both as it may handicap communication and as it occasions embarrassment. This disability may be exaggerated during illness and in circumstances of excitement. It is not to be expected that speech therapy will correct the basic disability of the pharynx, but it will promote greater skill in the use of retained abilities by training the patient to speak at a leisured pace, to enunciate clearly and to adapt to the embarrassment which may still result from his disability. In some subjects, it may be necessary to eliminate extraneous distortions of speech which tend to become habitual.

3. *Adequate care of intercurrent illness.* A characteristic occasion of the exaggeration of disability is an intercurrent illness, such as a respiratory infection. At such times, the patient who has partially recovered from bulbar-pharyngeal poliomyelitis may recapitulate the difficulties he experienced at an earlier stage of recovery. Thus the patient who has achieved a near-normal ability to swallow may be forced to return to a soft or a liquid diet.

The physician is particularly justified in attempting to abbreviate these intercurrent illnesses and to prevent their complication by adequate therapy. It is also recommended that these patients be thoroughly immunized against pertussis and diphtheria.

The persisting medical problems of the patient convalescent from acute bulbar poliomyelitis necessitates continuing medical assistance. This should include a routine of periodic visits in which the recent achievement of recovery and adaptation are reviewed. It is of considerable profit to the patient to evaluate and identify his disability with the aid of his physician, thus preventing both undue discouragement and execessive ambition to eat the more difficult solid foods, and to participate in general activity programs. The physician should watch for complications of residual paralysis, such as pharyngitis sicca.

SUMMARY

A series of 28 patients having persisting sequelae of disability following bulbar pharyngeal poliomyelitis has been reviewed. The basic patterns of pharyngeal paralysis and disability of function have been defined. The most common weakness is that of the levator muscle of the palate, which is associated with abnormal patency of the palatopharyngeal sphincter. The most important weakness is that of the pharyngeal constrictor muscles, which is associated with disability of swallowing. These patterns of weakness are inconstantly combined.

The criteria of identification of these forms of paralysis are described. The mechanism of associated disability of speech and swallowing and the resultant medical problems and therapy of the patients are discussed.

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LII

RHINOGENOUS INTRACRANIAL COMPLICATIONS IN THE ANTIBIOTIC ERA

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Severe intracranial complications of acute and chronic infections of the nose and paranasal sinuses are not uncommon. In some cases they may develop following surgical procedures, while in others there is extension intracranially unprovoked by operative manipulation. The complications we have observed and treated include extradural abscess, subdural abscess or empyema and brain abscess. The patients have been seen during the past two years in a period when chemotherapeutic and antibiotic drugs have been widely available and extensively used. Despite this, the nasal and sinus infections have been able to progress and extend to serious intracranial infections. Undoubtedly, in some cases adequate antibiotic therapy was not given early enough and maintained for sufficiently long periods of time. On the other hand, it is well known that chronic infections of the paranasal sinuses have not responded as well to the sulfonamides and antibiotics as have acute infections in these regions. Many of these infections may have deep-seated bony involvement. In addition, many may be caused by organisms which are very resistant to the antibiotics most commonly used. Regardless of the reason for the failure of the therapy to control the local infection, its spread to the vital structures within the cranial cavity presents additional problems to the otolaryngologist who must be able to interpret the symptoms

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which develop and to treat the complications as well as the underlying disease.

In all types of intracranial infections of rhinogenous origin four tissues are exposed to infection including the mucosa of the nose and paranasal sinuses, the bone, the meninges and the brain. Spread from the mucosa to the deeper structures may extend through the bone by contiguity or by continuity. In either case the soft tissues about the bone, the blood vessels, periosteum and endosteum, react first to the infection. A large number of anastomosing blood vessels, usually veins, run from the mucosa to the dura in the region of the posterior wall of the frontal sinus, the roof of the ethmoid sinuses, and the roof and lateral wall of the sphenoid sinus. As the infection spreads, periphlebitis about the walls of the veins or thrombophlebitis involving the lumen of the veins may develop producing bony involvement in the form of osteitis or actual involvement of the marrow, osteomyelitis.

Extradural abscess (pachymeningitis externa) may develop by contiguity or continuity. Spread by continuity along the blood vessels, through congenital dehiscences in the bone of the cribriform plate or along the sheaths of the olfactory filaments may occur but is not common. Rhinogenous pachymeningitis usually originates in an infection of the frontal sinus. This may travel through the posterior wall of the sinus by contiguity to produce an external pachymeningitis which commonly remains localized. In other cases the frontal sinus infection produces osteomyelitis of the skull which, in turn, may give rise to an extradural abscess. In most cases of extradural abscess of nasal origin, there is gross bony involvement, often found on the x-rays as osteomyelitic changes with or without evidence of sequestrum formation.

Subdural abscess, subdural empyema or pachymeningitis interna is an inflammation of the internal layer of the dura which usually produces a purulent exudate. In three of our cases pus was found beneath the dura, in one together with a small abscess of the frontal lobe. Commonly the infection travels by contiguity through the posterior wall of the frontal sinus or roof of the ethmoid with external pachymeningitis developing first. Spread may then occur along the dural blood vessels to produce an abscess beneath the dura. However, in some cases, infection may travel by way of anastomosing veins from the paranasal sinus mucosa to the internal layer of the dura without producing inflammation of the outer layer. In these patients the relatively normal appearance of the dura when exposed at surgery must not be permitted to prevent adequate search for the source of the symptoms.

Less commonly subdural abscess developing by continuity may occur with thrombophlebitis of the dural sinuses. Veins of the internal layer of the dura drain to the dural sinuses. With thrombophlebitis of the superior longitudinal sinus, infection may travel to the frontal lobe area and result in a subdural abscess without development of external pachymeningitis, and the outside of the dura appears normal at operation. Internal pachymeningitis is most frequently found at the base or on the anterior and lateral surfaces of the frontal lobe if it originates in the paranasal sinuses.

A third type of intracranial complication of rhinogenic origin is brain abscess. In most cases these are located in the frontal lobe. They are usually due to infection extending both by contiguity and continuity. In those cases extending by contiguity, gross changes in the bone and dura may be observed at surgery. When extension occurs by continuity, brain abscess may develop without significant abnormal findings observable grossly in the overlying bone and dura. Brunner states that in his experience, the bone was grossly normal in 70 per cent of cerebral abscesses. He also noted that there was no external pachymeningitis in 44 per cent of cerebral abscesses. Others have reported widely varying figures in this regard. Most important, is the fact that, as with subdural abscess, brain abscess may be present beneath bone which appears normal and beneath dura which appears normal. Further complicating the picture is the fact that following rhinogenous osteomyelitis of the skull, external pachymeningitis may develop beneath the involved bone and brain abscess may develop elsewhere beneath normal dura through spread by continuity.

Early, the infection of, the brain produces inflammation of the subcortical region by spread along the blood vessels from the leptomeninges. Later, there is breakdown of the infected area to form an abscess. The wall of the abscess consists either of necrotic brain tissue or of connective tissue with proliferation of glial cells. These tissues ultimately form a capsule around the abscess. Encapsulation does not cure the abscess, nor is it a static condition always preventing further spread. Frequently, necrosis of the capsule may involve the entire wall and permit purulent exudate to leak into the ventricle, subdural or subarachnoid space.

The time required for encapsulation varies widely and in some cases a well defined capsule is not found even after four to five weeks. If possible, delay in surgical treatment is advisable to permit adequate localization of the infection. Kahn gives several criteria to assist in determining the state of encapsulation. These include: (1) time—at least 10 days and preferably longer, (2) diminution of signs and

symptoms, and (3) a small number of lymphocytes in the spinal fluid in contradistinction to larger number of polymorphonuclear leukocytes.

There are no set rules in this determination and, unfortunately, the clinical course often demands surgical intervention before the optimal time is reached.

Our series of cases to be presented is small, but each case showed a clinical course and clinical findings which may be reviewed in some detail in order to correlate the symptomatology and progress of the disease with the pathological findings so that earlier and more accurate diagnoses may be made in future cases. These patients are usually desperately ill when admitted to the hospital and rapid surgical intervention is often necessary to save their lives.

CASE 1.—C. B., a 17 year old, Negro, high school student was admitted to the Neurosurgery Service of the Research and Educational Hospital, University of Illinois, on July 30, 1951, with a history of recent severe headache and convulsions. He was apparently well until July 14, 1951, when he developed a continuous right frontal headache. On the following day, the patient had projectile vomiting and chills. A physician made a diagnosis of sinusitis and prescribed capsules. On July 18, swelling of the right upper eye lid appeared. A rhinologist examined the patient on July 21, and confirmed the diagnosis of right frontal and maxillary sinusitis by x-ray. On July 23, nine days after the onset of symptoms, the patient suffered generalized convulsions originating in the upper right extremity. These were followed by periods of unconsciousness and recurred at frequent intervals. He was hospitalized elsewhere and had no further convulsive seizures during the ensuing seven days. Frontal headache, repeated bouts of projectile vomiting, and swelling of the right upper eyelid persisted. He ran a low-grade fever. Sedation and antibiotics were administered, and, following the development of nuchal rigidity, he was transferred to this institution.

The patient was a well-developed, well-nourished, Negro male, 17 years old, oposthotonic, with photophobia and nuchal rigidity. His temperature was 99, pulse of 64, respiration 24, and blood pressure 130/70. He appeared somnolent and irritable. His memory was poor and his ability to concentrate limited. He was disoriented as to time, but not as to place. He showed no aphasia or asterognosis. The right upper lid was moderately edematous, and there was tenderness over the floor of the right frontal sinus. The pupil was somewhat irregular and did not react to light. Blurring of the lateral margin of the right optic disc was noted. No apparent visual field

defect was present. The nasal cavities contained scant, dried mucopus, but the mucosa appeared normal. Nuchal rigidity and positive Kernig and Brudzinski signs were present. A left hemiparesis was evident and deep tendon reflexes were exaggerated, but bilaterally equal. No abnormal reflexes were elicited.

Hemoglobin was 15.5 gms per 100 cc, and the hematocrit 49. There were 5.2 million red blood cells per cu mm and 5,500 white blood cells per cu mm with a normal differential. Urinalysis and blood chemistries were normal. Wasserman and Kahn tests were negative. The spinal fluid was clear and colorless with a pressure of 300 mm H_2O. The Pandy test was negative. Seven lymphocytes per cu mm and 30 mg protein were present. No organism could be cultured.

By x-ray the skull showed no abnormalities. The right maxillary and frontal sinuses showed membrane thickening with several vague, soft densities in the right frontal compartment. There was no evidence of osseous involvement.

A diagnosis of right frontal sinusitis complicated by a right frontal lobe abscess and evidence of meningeal irritation was made. Because no specific organism was isolated, aureomycin in doses of 500 mg every six hours was administered. Neosynephrine was used to obtain shrinkage of the nasal mucosa. His temperature remained elevated until August 3, four days after admission. Irritability, nuchal rigidity and opisthotonos diminished and the left hemiparesis regressed. On August 7, while still on aureomycin, projectile vomiting recurred and swelling of the right upper eye lid reappeared. One diopter of papilledema was noted on the right, and abducens weakness was present on the same side. The patient remained afebrile.

On August 9, under local infiltration anesthesia, a burr hole was made in the right fronto-temporal region. Dilated vessels were visible on the surface of the dura. As the dura was incised, grayish, odorless, purulent material gushed forth. This was estimated to be about 60 cc. A ventricular needle was used to search for other collections of pus, but none was encountered. A soft rubber catheter was permitted to remain in the cavity. Cultures of the purulent material produced no growth.

Following the procedure, the patient became more alert and his irritability gradually disappeared. The drain was removed on the fifth postoperative day, and antibiotics were discontinued on the tenth postoperative day. He remained afebrile. A repeat lumbar puncture on August 21 revealed normal findings.

On August 29 three weeks following drainage of the subdural abscess, the right frontal sinus was explored through an incision made above and parallel to the right eyebrow. The anterior plate of the sinus was found to be softer and more vascular than is usual. The sinus mucosa appeared relatively normal, except for two areas of thickening. A portion of the posterior plate, which was intact, was removed exposing normal dura. Clear fluid was aspirated from the subarachnoid space. The patient made an excellent recovery, and has remained asymptomatic since his discharge from the hospital on September 4, 1951.

COMMENT

This patient showed rapid onset of symptoms and findings of an intracranial complication following acute right frontal sinusitis. There was no history to suggest that this was an acute exacerbation of a chronic frontal sinus infection. He had developed chills and projectile vomiting on the day after he had developed continuous right frontal headache. Presumably he was treated with capsules of one of the antibiotics.

X-rays a week after the onset showed right frontal and maxillary sinusitis. Nine days after the onset of his headache he had generalized convulsions. On admission to the Research and Educational Hospital he had marked rigidity of the neck, left hemiparesis, edema of the right uper end, and positive Kernig and Brudzinski signs. With this clinical evidence of severe meningeal irritation, there was increased spinal fluid pressure to 300 mm of water but otherwise the spinal fluid was practically normal, clear and colorless, with a negative Pandy, normal proteins and only seven lymphocytes per cu mm.

On antibiotic therapy in adequate amounts, symptoms and findings regressed only to recur with development of papilledema and right abducens weakness. Exploration revealed a large collection of pus beneath intact dura. Sinus surgery later showed grossly normal bone in the posterior wall of the frontal sinus and normal dura beneath it. The subdural abscess or empyema probably spread by continuity from a virulent acute infection of the mucosa of the right frontal sinus without producing bone destruction. At the time of admission to Research and Educational Hospital symptoms and findings were so severe and suggestive of localization that the beginning of an abscess must have been present. Spread was probably held in check by the antibiotics which could not cure the developing abscess. Without them, however, he might have gone on to a rapidly spreading suppurative leptomeningitis. A large amount of pus was present beneath practically normal dura, grossly normal bone and still the

spinal fluid showed only increased pressure and very few lymphocytes. Despite almost normal spinal fluid findings, the patient had clinical evidence of intense meningeal irritation. Marked neck rigidity and positive Kernig's sign are not common with subdural abscess unless spreading meningitis is developing.

CASE 2.—C.J., a 43 year old, white male plumber was admitted to the Research and Educational Hospital, University of Illinois, on October 3, 1951. He was irrational, disoriented and incoherent and his right upper eyelid was markedly swollen. His sister, who accompanied him, provided the history of his illness. For about nine years he had been treated intermittently for sinus trouble. About three months prior to his admission, he became withdrawn and paid little attention to his personal appearance. This change in his personality gradually became more marked. He began complaining of frontal headaches about one month prior to admission. One week after the onset of headache, a swelling of his right upper eyelid appeared and he was treated with injections of penicillin by his physician. The swelling subsided slightly, only to increase on the day of admission, as he became semi-delirious and his temperature rose to 103.

The patient was a well developed, well nourished, 43 year old male, who was markedly confused, irrational and incoherent. His temperature was 103, respiration 20, pulse 90, and blood pressure 135/80. There was a diffuse swelling and erythema of the right upper eyelid with a more discrete area of fluctuation on the outer espect of the region of the supraorbital ridge. External ocular movements were normal and both pupils reacted to light. Papilledema was not evident. There was a large spur on the left side of his nasal septum, scant pus was seen in the right middle meatus, and the nasal passages were adequate. The right maxillary and frontal sinuses did not transilluminate. Motor function was normal, but a valid sensory examination could not be done. The cranial nerves were apparently intact. Deep reflexes were normal bilaterally and no abnormal reflexes or nuchal rigidity were present.

The white blood count was 26,500 per cu mm with a normal differential, and the red blood count was 4.5 million per cu mm. The urine showed 4 plus albumin. Blood chemistries were normal. The spinal fiuid contained 8 polys per cu mm and the pressure was 280 mm H_2O. The Pandy test was negative.

By x-ray the right frontal, ethmoid and maxillary sinuses were cloudy. Active bone destruction and a large sequestrum was present in the right supraorbital region. A large spur on the left side of the septum was noted.

Following the spinal puncture, the patient was noted to have a left hemiparesis and to be semicomatose. Ankle clonus and positive Babinski sign appeared on the left, and superficial reflexes were absent on the same side. Because of the patient's critical condition, a frontal sequestrectomy, which had been contemplated, was considered too extensive a procedure for him to endure. Instead, under local anesthesia, a burr hole was made in the right fronto-temporal region. On opening the dura, about 40 cc of creamy, foul-smelling pus was released. The cortex in this region showed very little reaction of an inflammatory nature. The frontal lobe was tapped three times with a ventricular needle, but no other collection of pus was encountered. A soft rubber catheter was left in place to drain the subdural space and the wound was packed with petrolatum gauze. The right supraorbital abscess was incised, releasing 30 cc of purulent material. The patient immediately appeared more responsive following surgery. Cultures of the pus obtained showed no growth.

Following surgery, the patient was placed on aqueous penicillin, one million units every three hours, and intravenous fluids were administered. On the following day, he developed violent, left-sided convulsive seizures, which were difficult to control. Intravenous seconal was most efficient in managing them. His condition gradually improved, and by October 8, five days after surgery, he was practically convulsion free. His left hemiparesis regressed, and a spinal tap performed ten days later revealed normal findings.

On October 29, under endotracheal anesthesia, an incision was made along the upper margin of the right brow. A vertical midline incision over the forehead joined the medial end of the first incision, and a flap of soft tissue, including periosteum was elevated laterally and superiorly. This exposed a large sequestrum involving the right supraorbital margin. A dehiscence in the floor of the right frontal sinus led into the orbit. The macroscopically diseased bone plus a border of apparently normal bone one cm wide was removed. On exposing the dura, about 30 cc of purulent material escaped. The dura was dark in color, covered with a patchy, membranous exudate, and was necrotic in some areas. On incising the dura, a subdural collection of pus was released. The frontal cortex appeared injected and edematous. Tapping the frontal lobe to a depth of about 1 cm revealed another collection of pus, about 3 cc in volume. The dural incision was closed with interrupted sutures and a No. 18 rubber catheter placed through the frontal ostium for drainage. The skin flap was folded under itself and sutured in that position, leaving the wound open. A petrolatum gauze dressing was applied. Cultures of the pus revealed no growth.

He was placed on both streptomycin and penicillin, as well as anti-convulsant drugs. Except for an occasional seizure, he recovered rapidly, though he is maintained on dilantin and phenobarbital. To date, there is no radiologic evidence of residual or recurrent osteomyelitis. The forehead wound has been closed secondarily.

COMMENT

This patient, like the first had a subdural abscess but in addition he had osteomyelitis, extradural abscess and a small frontal lobe abscess. The course of his disease was quite different. He had a long history of sinus trouble. He had marked personality changes suggestive of frontal lobe involvement for several months prior to admission. There were no other localizing central nervous system findings until after lumbar puncture when left hemiparesis developed with ankle clonus and a positive Babinski on that side. His white blood count was markedly elevated to 26,500 whereas the first patient, though, also desperately ill had a 5,200 white blood count. Both patients had increased spinal fluid pressure with very few cells, normal protein, clear fluid and a negative Pandy test. This man, however, showed no stiffness of the neck, no Kernig or Brudzinski signs in marked contrast to the opisthotonus found in the first patient.

The pathognesis in this case appears to be spread intracranially by contiguity from chronic infection in the frontal sinus. Mucosa was involved first, later producing extensive osteomyelitic involvement of the bone followed by the extradural abscess formation and subdural abscess. The frontal lobe abscess was small and was beneath the cortex not simply cortical ulceration following a subdural abscess. Even though there was more extensive involvement in this case, there were fewer localizing brain findings on admission than in the first case. It seems reasonable to conclude that in acute fulminating infections there may be much more extensive encephalitis and brain edema with consequent findings than in chronic infections where spread occurs more slowly. In acute cases which are rapidly progressive findings may be on the basis of intense encephalitis rather than abscess and evaluation of the case surgically must consider this possibility. On the other hand, in chronic cases there may be extensive abscess formation with far fewer localizing findings to guide the surgeon.

CASE 3.—C. C., a 30 year old, white male stationary engineer was admitted to the Research and Educational Hospital, University of Illinois, on November 11, 1951, with a history of recurrent left frontal headaches for one year and vomiting for three days. In February, 1951, his headaches became severe, but were relieved by treatment for sinusitis. This consisted of injections of penicillin and

intranasal shrinkage. The left frontal pain, in a mild form, recurred periodically with intermittent nasal obstruction. In September, 1951, the patient underwent a right intranasal ethmoidectomy and a left nasal polypectomy, elsewhere. Following this procedure, the left frontal pain became more severe than it had ever been. He was again hospitalized by his physician and treated for a possible osteomyelitis of the left frontal bone. He became asymptomatic and was discharged in one week. Three days prior to admission to this institution, severe left frontal pain again recurred and vomiting without nausea developed. These symptoms had become progressively more severe. There was no history of personality, visual, sensory or motor changes.

The patient was a well developed, well nourished, 30 year old male, who appeared to be in moderate distress. His temperature was 98.6, pulse 60, respirations 16 and blood pressure 118/80. There were no apparent facial deformities. Both nasal cavities contained small amounts of mucopurulent material and polypoid tissue was present in the roof of the left nasal cavity. The frontal and maxillary sinuses did not transilluminate. The floor of the left frontal sinus was tender to palpation. The pupils were round and reacted to light normally. There was a suggestion of haziness of the margins of the right optic disc. Neurological examination revealed no abnormalities.

The hematocrit was 70 (though there was no clinical evidence of dehydration) and there were 15,000 white blood cells per cu mm. Urinalysis and blood chemistries were normal. With the patient in the right lateral recumbent position, a spinal tap was performed. The initial pressure was 370 mm H_2O, which in a few minutes dropped to 350 mm, but promptly returned to 370 mm. The fluid was crystal clear and a small amount was obtained for a cell count and Pandy test. The latter was negative and there were 6 lymphocytes and a polymorphonuclear cell per cu mm.

By x-ray all the sinuses appeared dense. In addition, the margins of the frontal sinuses were ill-defined and they were surrounded by a wide area of sclerosing osteitis. There was a suggestion of a sequestrum in the left supraorbital region.

A diagnosis of osteomyelitis of the left frontal bone, secondary to frontal sinusitis, and complicated by either a subdural or frontal lobe abscess was made. The patient was placed on one million units of aqueous penicillin every three hours and streptomycin, 0.5 gm twice daily. On the following day, under endotracheal anesthesia, the left frontal bone was exposed through an incision made along the upper border of the left brow and exending down to and across

the root of the nose. The anterior plate of the sinus appeared dull gray in color. The sinus lining appeared normal. A portion of the posterior plate of the sinus was removed, exposing an area of dura about 2.5 cm in diameter. The dura was a glistening white, but bulged through the defect in the posterior plate. An No. 18 needle was passed through the dura into what was thought to be the subdural space, but nothing could be aspirated. Again the needle was inserted to a depth of about 1.5 cm. Here, 15 cc of thick, gray, odorless, purulent material was obtained. A linear incision about 0.5 cm long was made through the dura revealing the bulging to be due to edema of the left frontal lobe. Nothing further was done surgically, except to pack the wound loosely with petrolatum gauze and permit it to remain unsutured. Cultures revealed an unidentifiable organism, probably Streptobacillus sensitive in vitro to 0.08 units of penicillin per cc and 5.0 units of streptomycin per cc.

Penicillin and streptomycin were continued in their previous doses. During the first two post-operative days, the patient was semi-comatose and his pulse remained between 56 and 60. Late on the second day, definite one plus papilledema was present and positive Kernig and bilateral Babinski signs were present for the first time. Believing these findings to be the consequence of cerebral edema, conservative therapy was continued. During the following week, his temperature remained between 99 and 100.4, he was conscious, but confused and disoriented. On the ninth post-operative day, the pathological reflexes and nuchal rigidity were no longer present. The dressings were changed every third day, revealing the depths of the wound to be free of purulent material and the protruding portion of edematous cortex to be receding. His sensorium gradually became more clear, and his improvement was such that on the eighteenth postoperative day, he was permitted to be ambulatory and antibiotics were reduced to 600,000 units of procaine penicillin daily. One month following surgery, a spinal tap revealed the spinal fluid to be under 200 mm of water pressure. The Pandy test was negative, and there were no cells. The patient remained asymptomatic, his wound closing by secondary intention, and he was discharged from the hospital forty days after drainage of his frontal lobe abscess. Follow-up clinic visits reveal him to be enjoying apparent good health and he has returned to work.

Reconstruction of the pathogenesis of the brain abscess in this case is not as clear as in the preceding cases. It may have developed following the intranasal surgery in September by continuity through venous channels in the nose and ethmoid region; or it may have followed a bout of frontal sinus infection several weeks after the surgical

procedure. However, that may be, it indicates emphatically that a patient with persistent localized headache must be carefully and repeadely observed over a long period of time despite the absence of neurological findings. Three weeks prior to the onset of vomiting, mild papilledema, and increased spinal fluid pressure, he had recurrence of his severe left frontal headache. Careful, detailed neuroalogical examination revealed no abnormal findings. There is little doubt that pathological changes had already begun at that time in a relatively silent area of the left frontal lobe. It is probably fortunate that symptoms and findings demanding surgical intervention did not occur early so that there was time for the abscess to become walled off before it was drained.

This abscess was approached through the frontal sinus which appeared diseased on the x-rays to treat the primary site as well as the intracranial complication. When this can be done safely, it is the procedure of choice. In the preceding case, the patient was so ill that extensive surgery through the sinus was considered to be more than he could tolerate. A less extensive operation, a burr hole, was the procedure of choice, to approach the abscess. Shuster, in a recent article on brain abscess stated "One hears of an otolaryngological approach and a neurosurgical approach as if they were two good methods and one has to make a choice. To my way of thinking there is no such thing as a choice." We do not believe that the approach should be considered in terms of the specialties. The primary consideration should be the safety of the patient. In our small series we have found that there may be a choice based on the welfare of the patient, and he must be given the benefit of the procedure which can be safely tolerated in certain instances.

COMMENT

There are three general methods used to treat brain abscesses:

1. Closed method: Once the dura is exposed a small opening is made in it and the edges are sealed. A blunt needle is introduced to palpate for the abscess wall and the abscess is aspirated. Penicillin solution may be instilled. The wound is packed open and later aspiration may be necessary. In some cases a small catheter may be placed into the abscess cavity.

2. Open method: The same approach is used but the bone opening is enlarged. The dura is opened with a stellate incision and sealed with electrocautery. Cortex is then removed exposing the abscess wall and the lid of the abscess is removed. The cavity may be irrigated with constant suction. It may be packed open with iodoform gauze.

3. Complete extirpation: This may be done in treating well delimited abscesses easily available surgically. It is reported to be more dangerous for large abscesses and those with a poor capsule. It cannot be used to treat deep seated abscesses without damaging a great deal of brain tissue. Davis states, "In our experience a spreading encephalitis is likely to develop from an attempt to remove the capsule."

In this case we used the closed method, aspirating the contents of the abscess cavity. There was marked bulging of the edematous cortex through the dural opening. Postoperatively the patient remained semi-comatose for over 48 hours. It was felt by some that this was the result of inadequate drainage of the abscess. Apparently it was due to the marked cerebral edema which increased postoperatively and may not subside for 48 to 72 hours.

CASE 4.—W. M., a 16-year-old Negro male was admitted to the Neurosurgery Service of the Research and Educational Hospital, University of Illinois on March 8, 1952. He was in good health until fourteen days prior to admission, when he returned home from a track meet with the "sniffles." During the following two days, his "cold" became more severe, he began to complain of pain behind both eyes, and he developed a small swelling over the glabella. In addition, twenty-four hours later, his right eye swelled shut. His physician diagnosed acute suppurative frontal sinusitis on the right and hospitalized him. His temperature ranged from 100.4 to 101.6. Therapy consisted of intramuscular penicillin, terramycin orally, and ice packs to the forehead. The patient improved, his temperature returned to normal, the swelling of the right eyelid disappeared, and he became ambulatory. The swelling over the glabella, however, became larger and softer. Sinus x-rays taken on three occasions during this period were reported as normal.

The patient was doing well until the morning of March 7, 1952, when he collapsed while trying to get out of bed. It was noted that a right hemiplegia was present and that the patient had become semi-comatose. A spinal tap showed the fluid to be under increased pressure and to contain 1600 white cells per cu mm, 75% of which were polys. The Pandy test was positive. In addition to the antibiotics already being administered, sulfadiazine, 10 gm every twelve hours, was administered intravenously. Later that day, the patient began to have twitchings of the right leg, and on the following day, he had frequent intermittent grand mal seizures involving the entire body.

The patient was a well developed, well nourished, adolescent Negro male, appearing acutely ill and evidencing a complete motor

aphasia, a right hemiplegia, and almost constant convulsive activity of the right lower extremity. His temperature was 100.2, pulse 76, respirations 20, and his blood pressure was 130/50. A soft, fluctuant mass, about 3 cm in diameter was present over the glabella. Fundoscopic examination revealed the optic cups to be obliterated with distention of the retinal veins. A severe right facial paresis was present. No purulent material was noted in the nasal cavities. Moderate nuchal rigidity and bilateral Kernig signs were present. The deep tendon reflexes were equal on both sides and plantar reflexes were normal. Reaction to pin pricks was equal on both sides. The frequent seizures began with convulsive movements of the right side of the face, followed by about 60 seconds of convulsive activity of the right side, then both sides of the body. During the seizures, the head and eyes turned toward the right.

By x-ray examination there was clouding of the left frontal and ethmoid sinuses. No evidence of bone destruction was present.

The abscess over the glabella was aspirated of 3 cc of thick, grayish purulent material, which showed no organisms on stain or culture. The patient was placed on penicillin, one million units every two hours, aureomycin, 500 mg every four hours, and gantrisin 1.0 gm every four hours. Intravenous sodium amytal was administered to control the frequent grand mal seizures. The patient's sensorium became less clear as the convulsions continued. On the following day, he developed Cheyne-Stokes respiration and the left pupil became dilated and fixed. He expired about twenty-four hours after admission.

At postmortem examination there was found a thick, purulent, exudate in the subdural space extending from the left orbital region around the frontal pole and over the surface of the left hemisphere as far back as the parieto-occipital fissure. No purulent material was present in the basilar cistern. The exudate extended over the medial surface of the hemisphere as far as the corpus collosum and behind the pineal body onto the undersurface of the occipital lobe. Purulent material was present in the frontal, ethmoid and sphenoid sinuses. Osteomyelitic changes were evident in the frontal bone.

COMMENT

This is another case of acute frontal and ethmoid sinusitis with a rapid course producing a fulminating, purulent meningitis which did not become well localized and was not controlled with antibiotics. He had received antibiotic therapy including penicillin and terramycin and intravenous sulfadiazine. In contrast to the first two

patients who had subdural abscess with clear spinal fluid and a negative Pandy test, he had a cloudy fluid containing 1600 cells, chiefly polys and a positive Pandy test. This patient had pus in the subdural space but all therapy failed to limit the infection and spread to the leptomeninges followed. Though x-rays repeated several times prior to admission to our hospital were reported as normal, our films revealed clouding of the left frontal and ethmoid sinuses. Postmortem examination showed pus in those sinuses and revealed gross evidence of osteomyelitic changes in the frontal bone which had not been demonstrated on the x-ray films.

CASE 5.—J. O., a 36 year old white male was admitted to the Veterans Administration Hospital, Hines, Illinois, on January 21, 1952 with complaint of frontal headaches, present for about two weeks prior to admission. In the last few days before entrance into the hospital, these became appreciably more intense and were more marked on the left side. The history was elicited with great difficulty. The patient was cooperative but he apparently was unable to answer questions satisfactorily.

Examination revealed a well nourished, well developed acutely ill, white male. Temperature was 102.6, pulse 88 and blood pressure 114/66. The right pupil was slightly larger than left. There was mucopurulent discharge in the nose bilaterally, but no obstruction. The ears appeared normal. Frontal and maxillary sinuses were dark on transillumination. There was tenderness over both frontal sinuses. The neck showed slight rigidity when anteflexed. The remainder of the physical examination was non-contributory. Neurological examination was negative except for the difference in the size of the pupils and nuchal rigidity. Diagnosis of acute left maxillary and frontal sinusitis, with possible intracranial extension was made at this time.

Two days after admission the patient developed a motor aphasia, with a total vocabulary of one word, "No," to all questions. The temperature rose to 104. Examination of the spinal fluid at that time showed 18 WBC per high power field, 41 mgs of sugar, 60 mgs total protein, Wasserman was negative and the colloidal gold curve was 0012110000. Neurological examinations revealed absent abdominal reflexes, nuchal rigidity and aphasia. There was no papilledema. The impression of the Neurological Department was that this was a meningitis. The patient had been on penicillin, 300,000 units a day, and at that time streptomycin and gantrisin were added in therapeutic doses.

Examination note by the Neurological Service on January 24, 1952, was as follows: "Patient seems brighter, temperature down to 100.2. The aphasia remains. The left pupil is still smaller than the right; they both react to light. The left palpebral fissure is noted to be narrower than the right. The cranial nerves are otherwise intact. There is no ataxia on finger-to-nose test. The knee jerks are hyperactive, the right being slightly greater than the left. The ankle jerks are present and equal. The left plantar reflex is normal. The right shows a quick dorsiextension of the big toe and dorsiextension of the other toes. There is no positive Gordon, Chaddock or Oppenheim sign. The abdominal and cremasteric reflexes are equal and lively. Slight nuchal rigidity remains. No Brudzinski sign. There is a positive Kernig sign on the right. Impression: This man still has signs of meningeal irritation with a possible left Horner's syndrome; brain abscess must be ruled out."

At 8:30 p. m. on January 24, 1952, the patient had a convulsion and when seen by the ward physician he had a right hemiplegia. He was sedated and slept well all night until 5:30 a. m. which time he had a second but slight convulsion. The right arm showed clonic movements. The patient did not lose consciousness. Examination in the morning revealed that he was still aphasic but tried to cooperate. The second spinal fluid examination showed no cells and 30 mg of protein. Nuchal rigidity and bilaterally positive Kernig signs were still present. The right nasolabial furrow was not as deep as the left. There was falling away of the right outstretched arm. The patient did not move the right leg and a right Babinski sign was noted. This examination showed a right hemiplegia with a central facial paresis. It was thought he had a brain abscess in the left motor area or extra- or subdural abscess.

The next day the patient was taken to surgery and under general anesthesia the frontal sinus was opened through an incision through the left eyebrow and vertically up the midline of the forehead. The frontal plate appeared intact. The sinus was opened and purulent material was immediately found and aspirated. The entire frontal plate was removed and the posterior plate was examined. This was found to be intact and composed of apparently solid bone. Nevertheless, this was taken down exposing dura. The dura appeared normal but a No. 22 needle was placed through the dura to examine the contents of the subarachnoid space. No evidence of infection was found. A rubber catheter was left in place for drainage within the sinus and the wound edges were approximated with silk sutures. On the left maxillary sinus a Caldwell-Luc operation was performed and purulent material was found far posteriorly in the antrum. The

lining was completely removed by curettage and a window was made into the left inferior meatus.

The patient had a convulsive seizure on the night following surgery. It was generalized but more prominent in the right upper and lower extremities. During this seizure the head was turned to the right and the eyes were deviated to the right. The convulsion was controlled with intravenous seconal. The following night the patient had a similar seizure which lasted only a few minutes.

By January 28, three days after surgery, the aphasia had cleared but the patient was still febrile and had been ever since surgery. He was able to carry on a conversation but was unable to move his right leg. He had full use of his arm although weakness there was noted. The next day function of the leg began to return. However on that day visual field examination revealed a decrease from the normal periphery in the left eye with an indenting defect 10° from the point of central vision.

On January 30, 1952 there was no elevation of temperature and there has been none since.

Examination on February 5, 1952, revealed that the patient was well oriented and in good contact and had regained almost complete function of his right arm. A difference in pupillary size still remained. The knee jerks were active and the right was greater. There was no Babinski, but there was a right ankle clonus. The right heel-to-knee test was ataxic.

Until three weeks following surgery, the patient was able to take no more than two steps without falling. At that time, he was found to be able to walk up a small flight of stairs with little assistance. The function of the right arm is apparently completely recovered. The right leg improved steadily.

COMMENT

This case is particular of interest in contrast to the other cases cases presented because there were localizing central nervous signs strongly indicating the presence of a subdural abscess or left frontal lobe abscess but at surgery none was found. Needle exploration of the left frontal lobe was not done when the surgeon found the dura and subdural space showed no abnormal findings. On retrospect this exploration should have been accomplished when no abscess was found beneath the dura to account for the right hemiplegia and convulsive seizures. There is very slight risk to such an exploration. There is great risk in leaving an abscess untreated. One risk should be

weighed against the other and the surgical management in part determined by the result. Frontal lobe abscess may be present in the absence of bony involvement, when the dura is normal in appearance and with spinal fluid findings which show no cells and a normal protein level.

Postoperatively, this patient showed progressive improvement after a short febrile period during which he had two more convulsive seizures. It seems most likely that the localizing symptoms were caused by an acute fulminating sinus infection producing meningeal irritation manifested by stiffness of the neck with encephalitis involving the left frontal lobe. There has been no recurrence of symptoms during the past year.

SUMMARY

Widespread use of antibiotics and chemotherapy has not eradicated severe intracranial complications of acute and chronic infections of the nose and paranasal sinuses. The otolaryngologist must be able to recognize and interpret the neurological symptoms and findings and to treat the primary focus and the complications. The neurological manifestations do not always conform to a classical pattern. These case reports have been presented for whatever aid they may provide in the management of such patients in the future.

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LIII

PNEUMOTHORAX AS A COMPLICATION OF AN INTRABRONCHIAL VEGETABLE FOREIGN BODY

REPORT OF A CASE

WILLIAM BLAKE HARKINS, M.D.

ST. LOUIS, MO.

Pneumothorax resulting from the aspiration of a foreign body into a bronchus is of sufficiently unusual occurrence to warrant the reporting of a case. It is not intended to include in this discussion instances resulting from aspiration of a sharp-edged or pointed foreign body, which mechanically tears the wall of the tracheobronchial tree, nor those obviously resulting from perforation of the tracheobronchial tree by a bronchoscope, nor from tracheotomy performed after foreign body aspiration.

A review of the literature for definite case reports yielded only seven instances of pneumothorax occurring under these circumstances. Clerf[2] in a report of 413 cases of foreign body in the air passages had one instance of pneumothorax of slight degree, following the aspiration of a bean. The pneumothorax subsided without further treatment, after bronchoscopic removal of the bean. However, a tracheotomy became necessary after bronchoscopy because of laryngeal edema.

Holinger, Andrews and Anison[3] reporting their experience with 35 tracheal foreign bodies and 318 bronchial foreign bodies had one instance of a slight pneumothorax, with minimal collapse of the lung, from a tooth in the right upper lobe bronchus. Following removal, the air in the pleural space gradually resorbed without further complication.

Kaludjerski[6] reported an instance in a fourteen-month old baby of an aspirated orange seed with an obstructive pulmonary emphysema in the affected side. Four days later pneumothorax was discovered on this same side. The orange seed was coughed out spontaneously four days later; thereafter the pneumothorax subsided without further treatment.

From the Department of Otolaryngology, Saint Louis University School of Medicine, Saint Louis, Missouri.

Cantorné and Despons[1] reported a case, in an eleven-year-old girl, of pneumothorax and collapse of one lung after aspiration of a foreign body. At bronchoscopy the foreign body was not discovered, but on the fifth postoperative day an object of undetermined nature, twelve millimeters long, was coughed out; thereafter the air in the pleural space was resorbed and the lung re-expanded.

Kaludjerski[5] had another case in the literature. This was a two-year old child who aspirated two roasted coffee beans. One was coughed out, but the other bean remained in the left main bronchus. There was severe dyspnoea; a tracheotomy was performed. It would appear that in this case the pneumothorax may have resulted from the tracheotomy.

Rubin[7] in his textbook on diseases of the chest relates one case-history: a two-year old child who had aspirated a peanut. Twelve hours later there was widespread emphysema of the subcutaneous tissues of the neck and chest, in the muscle planes of the neck, thorax and abdomen and in the mediastinum. Several hours later the child was found to have developed right-sided pneumothorax. After removal of the foreign body the emphysema and pneumothorax subsided spontaneously. In discussing this condition Rubin makes the statement that foreign body aspiration is one of the major causes of spontaneous pneumothorax in infants and children, and that there is an interstitial pulmonic emphysema. The partial occlusion of the major bronchi results in the so-called "obstructive emphysema." The rapid increase in pressure causes subpleural alveolar rupture, resulting in spontaneous pneumothorax.

Jackson and Jackson[4] describe one case in a fourteen-year old child who had aspirated a cockleburr seven months earlier. This child had a large pyopneumothorax which was drained by external thoracotomy. Two months later, when the patient's condition had improved, the foreign body was removed bronchoscopically. There was an inflammatory stenosis of the bronchus which was completely occluded. No bronchopleural fistula could be demonstrated. In discussing this subject Jackson and Jackson state that in most instances pneumothorax in foreign body aspiration cases is caused by the trauma of instruments. However, they go on to say that in some cases pneumothorax has occurred in patients on whom there had been no instrumentation. In the Jacksons' clinic, in a number of such cases, pneumothorax was associated with subcutaneous emphysema, and with mediastinal emphysema. These cases were in the nut-kernel group, and associated with obstructive pulmonic emphysema. Mention is also made of instances due to tearing by sharp-edged or pointed objects.

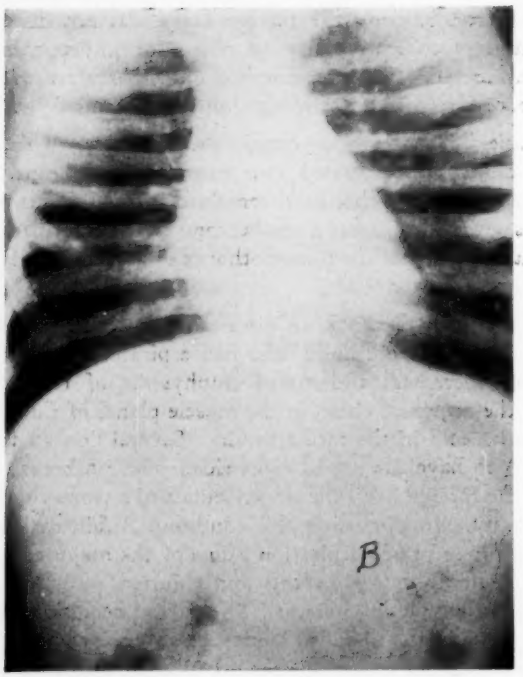

Fig. 1.—The chest immediately prior to bronchoscopy, showing right-sided pneumothorax with partial collapse of the right lung.

Our own experience is limited to one case.

REPORT OF A CASE

The patient was an eighteen-month old boy. The history is this: that while eating a candy-bar covered with chopped pecans the child was observed to be choking. He coughed, gagged and had difficulty in breathing for a few minutes. Thereafter, the respirations became effortless and the child seemed comfortable except that there was audible wheezing. Approximately twelve hours later an x-ray picture of the chest was taken in another city (this film was not available to us), which was said by the local medical doctor to have shown atelectasis of the right middle lobe.

The child's past history and family history were not contributory to the present illness.

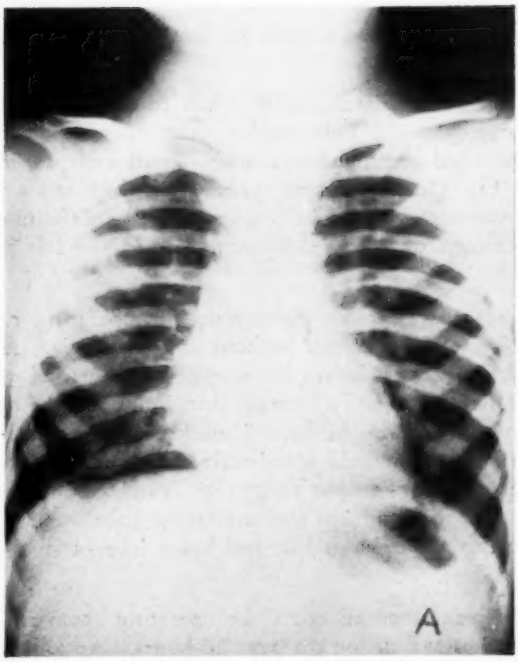

Fig. 2.—The chest approximately twelve hours after bronchoscopy, showing partial resorption of the right-sided pneumothorax.

The patient entered St. Mary's Hospital on Sept. 9, 1949, and was seen approximately two hours after the first x-ray picture of the chest had been taken. Physical examination on admission to the hospital revealed a well-nourished, well-developed male child, crying lustily. The voice was clear; no hoarseness was noted. There was audible wheezing. Slight cyanosis of the skin was noted. Examination of the chest revealed slight inspiratory retraction of the soft tissues (suprasternal and supraclavicular) and the lower ribs, but it was apparently equal on the two sides. Percussion revealed hyperresonance in the right axillary and the anterior aspect of the right side of the chest. There was normal resonance elsewhere over the lung fields. Auscultation revealed very faint and distant breath sounds in the right axilla, over the right anterior chest and the right base, with very faint inspiratory and expiratory dry wheezing râles over the entire right chest. The breath sounds over the left chest

were loud, rough and accentuated, but no râles were heard on the left side. The area of deep cardiac dullness, heart sounds and the apical impulse were in the normal location. Other physical findings were essentially negative.

The patient was immediately taken to the x-ray department, where fluoroscopic and radiographic examination of the chest revealed right-sided pneumothorax with partial collapse of the right lung (Fig. 1). On fluoroscopic examination there was some restriction of movement of the right diaphragm and a definite pendulum-like displacement of the mediastinum toward the left side during expiration.

The infant was taken directly to the operating room where bronchoscopy was carried out without any anesthesia. A 5 mm by 30 cm Jackson Jackson infant bronchoscope was passed without any apparent difficulty. The right main-stem bronchus, the right middle and the lower lobe bronchi showed marked swelling and redness of the mucosa, and contained blood-stained mucus and fragments of nut in the lumen. The nut fragments crumbled into bits as they were extracted with forceps and aspirating tube. Air appeared to be passing into the right middle and lower lobe bronchi at the end of the procedure.

The patient returned from the operating room in good condition and remained so for the next 24 hours. An x-ray picture of the chest the morning following bronchoscopy (Fig. 2) revealed partial absorption of the air previously present.

Twenty-four hours after the bronchoscopy the infant's breathing became labored and within the next hour he developed typical laryngeal stridor, the voice became hoarse, and mild cyanosis developed; he became increasingly restless and the respiratory rate was rapid (48 per minute). It was felt that the child had developed an acute larygeal edema as a result of the bronchoscopy. Tracheotomy was performed and a No. 3 tracheal cannula was inserted with immediate relief of the dyspnoea. We were able to decannulate the patient six days later. The child left the hospital five days later in good condition, the right lung completely re-expanded.

3720 WASHINGTON BLVD.

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LIV

A NASOPHARYNGEAL SPECULUM

BEN H. SENTURIA, M.D.

ST. LOUIS, MO.

During the past three years we have searched the literature for more efficient techniques or better instruments for visualizing the less accessible areas of the nasopharynx. A search of available information disclosed the early views and measurements of von Gyergyay.[1] Shortly thereafter Yankauer[2] presented the speculum which bears his name. Then Beck[3] described the use of a rubber catheter passed through the nasal fossae for retraction of the soft palate. Some years later Kelly[4] described his ingenious direct vision adenotome. Subsequently there appeared a note by Love[5] detailing a palate retractor which has received wide usage and Zollner[6] published interesting diagrams and a discussion of the Gyergyay instruments in his monograph. Instruments designed by Lathrop and Cottle are available but no references in the literature could be found. A combined mouth-gag and adjustable palate retractor was described by Feinberg[7] and a self-retaining illuminating palate retractor by Dalton.[8]

Each one of these instruments has very definite merit but in some manner or another has limitations which restrict its use. With most of these instruments, there is difficulty in exposing the roof of the nasopharynx, the pharyngeal recesses and the tubal folds. The Yankauer speculum, in particular, has a relatively small lumen which restricts visibility and prevents the free use of suction and biopsy forceps.

In preparing drawings and models of a projected instrument, cognizance was taken of the available anatomical measurements and models of Escat[9] and the excellent descriptions of Blumenthal.[10] Due consideration was given to the dicta stressed by von Gyergyay.[1] Thus it was considered desirable for the curvature of the instrument to fit the concavity of the posterior margin of the hard palate. The

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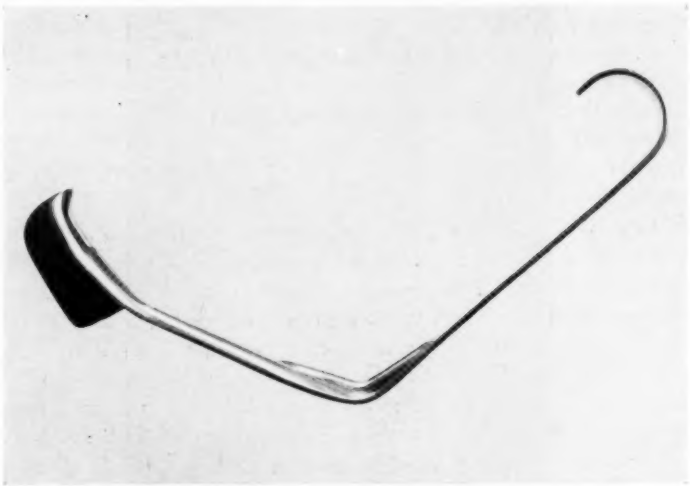

Fig. 1.—Side view of nasopharyngeal speculum showing long, flat, angled handle to which is attached an inverted incomplete U-shaped Tube. Note the absence of restricting walls.

posterior nasal spine was to be avoided. The soft palate and uvula should lie flat on the superior surface of the instrument. If possible, the distal end of the instrument ought not to project very far above the hard palate. There must be sufficient inferior and lateral protection to prevent obstruction of the view by the tongue and sides of the mouth.

In the first drawings of the instrument, a light carrier and a tubular suction were included very much as found on an esophagoscope. It developed, however, when models were prepared, that the large unobstructed lumen permitted adequate illumination from a reflected light source permitting the removal of this expensive and potentially troublesome appendage. When it was demonstrated that very adequate suction could be obtained through the speculum with the standard suction apparatus, the attached suction tube was omitted.

The most recent model of this simple nasopharyngeal speculum consists of a long, flat, angled handle to which is attached an inverted incomplete U-shaped tube (Figs. 1 and 2). It has an upturned flanged tip to facilitate introduction into the nasopharynx and to assist in the retraction of the soft palate. The speculum is made of

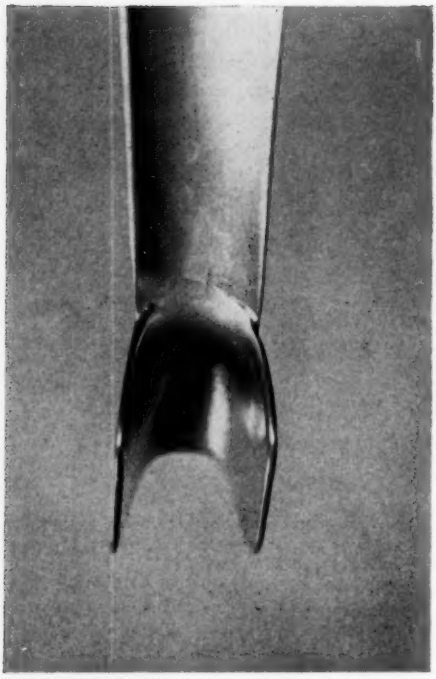

Fig. 2.—Nasopharyngeal speculum viewed from above and showing excellent visibility.

hard brass and is plated. At the present time it is available in one size for use with children. Another model for adults is being tested. Other sizes may be necessary.

It is suggested that the instrument be used prior to and following the removal of the adenoids with the adenotome or curette. The patient's head is placed in a slightly extended position to prevent drainage of blood into the laryngo-pharynx. The tongue is pulled forward and downward with the aid of a towel clip attached to the superior surface of the tongue. The upturned tip of the speculum is then inserted under the free border of the soft palate and with gentle traction the soft palate is "bulged" forward and upward.

Residual adenoids far anterior on the roof, hanging down as a curtain over the posterior nares may be visualized and removed easily.

The adenoid extensions into each pharyngeal recess may be successfully seen and nibbled away with an instrument like the Meltzer or Guggenheim punches.[11] The hyperplastic lymphoid tissue on the salpingo-nasal and salpingo-palatine folds may be exposed readily after a little experience with the speculum. Finally, with good light and suction available through the speculum, every part of the nasopharynx may be carefully inspected for bleeding sites. The source of any persistent bleeding may be isolated and evaluated. Thus it is possible for the surgeon who insists on a bloodless nasopharynx to be assured of a clean, dry field when the patient leaves the operating room table.

Appreciation is expressed to Mr. Eric Storz and Mr. Herman Belz of the Storz Instrument Co., St. Louis, Missouri, and William Breghoff for technical assistance in preparing the speculum.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 5, 1953

THE PRESIDENT, DR. LAWRENCE J. LAWSON, IN THE CHAIR

Rhinogenous Intracranial Complications in the Antibiotic Era

EMANUEL M. SKOLNIK

BURTON J. SOBOROFF

ROBERT B. LEWY

AND

FILMORE SCHILLER (By Invitation)

(Complete paper appears on page 548)

DISCUSSION

DR. SHERMAN SHAPIRO: This is a splendid group of cases and the one which interested me especially was the one in which the frontal sinus was opened after the brain abscess had been operated on and was found to be normal; the assumption was made that there had been a subdural abscess and that the situation had occurred as a result of continuity. I think this case is a fairly typical example of retrograde simultaneous infection of the brain and the frontal sinus, the same type of condition reported from year to year, following swimming, for instance, where the individual has or has not a cold and the cerebral symptoms and the frontal sinus symptoms have been practically simultaneous. We can expect such occurrences regardless of antibiotics; antibiotics will not prevent them because the organism or infection is in the brain before the antibiotic has a chance. Th brain is avascular and no antibiotic can reach it.

In the other cases the origin can be disputed in various ways, but this particular method of invasion will always be with us, and

should always be kept in mind in any acute or fulminating rhinogenic or otogenic infection.

DR. SAMUEL SALINGER: This paper is reminiscent of the past; the days when I first joined this Society, when intracranial complications were quite common. The statement was made that antibiotic therapy was employed. The question arises as to how effective it was, how much was administered and how specific it was in relation to the bacteria. In classifying these complications, I have thought that in the acute cases complications are more likely to be by way of retrograde thrombophlebitis, with a minimum of local change in the sinuses; whereas in chronic cases with acute exacerbation, we are more likely to find changes in adjacent bone and other tissues. Consequently much depends on the history of the case. In my opinion a chronic case with acute exacerbation, irrespective of whether or not there are focal signs, would demand exploration of the sinuses; whereas in some cases, particularly the acute fulminating type, the theory has been to go directly toward the focus in the brain. At any rate, these cases have proven that antibiotics perhaps are not the last word in prevention of these complications.

DR. O. E. VAN ALYEA: This is a highly important subject, and it is rather surprising that we do not have more reports of intracranial complications of sinus origin. I have seen some of these cases that were reported during the past year or so at the Research Hospital in our seminar. I noticed that in most cases patients had received antiobiotic therapy prior to examination by an otolaryngologist. If we learn nothing more from this presentation, we learn that we cannot be too emphatic in decrying the use of antibiotics in early cases of sinusitis. It is far more important to have a proper diagnosis and proper evaluation of the organisms before the drugs are used, and thorough study with the idea that they should be withheld until their use will be greater value; that is, when there are threatening intracranial complications. We also learn, regardless of the fact that antibiotics are so highly important in many acute infections, that there is no substitute for drainage of closed-off cavities.

DR. T. C. GALLOWAY: This very beautiful presentation does show that what we thought was a closed chapter in otolaryngology may have to be reopened because of the development of resistance of organisms to antibiotics. I was surprised that in the study of all these cases there was no report of bacteriologic studies. Things come as new every once in awhile that we thought we had studied out pretty well a long time ago. Furstenberg and Mosher and others worked out the pathogenesis and progress of osteomyelitis. Some

of these were retrograde infections. There were in the terminal stage certain types of osteomyelitis definitely known to be associated with Staphylococci.

I reported thirteen consecutive cases of intracranial complications in each of which we were able to find micro-aerophilic or anerobic Streptococci, which I believed to be the primary invader. One is rather justified in assuming that in the case reported, in which much foul pus was obtained, there was an anaerobic infection. The anaerobes in the more chronic lesions prepared the way with a thrombophlebitis, followed by a secondary infection with Saphylococcus, which progressed in large part at least by a spreading thrombophlebitis. If we say the patient did receive antibiotic therapy, it seems to me we must know it was an organism in which the particular antibiotic is effective. We should have sensitization tests and we should know that the antibiotic is used in adequate dosage. We know, for instance, that the anaerobic oragnisms did not respond at all to the sulfonamides, but they did respond very well to penicillin and we got much better results after penicillin was introduced. Much attention is now being paid to the fact that certain strains of Staphylococci are becoming resistant. In a certain New York Hospital it has been found that five years ago 70 per cent of the strains of Staphylococci were sensitive to penicillin; now 30 per cent are. We might even have recrudescence of these much to be dreaded intrachanial complications.

Dr. B. J. Soboroff (closing): I should like to thank the discussors for their comments. Cultures were made of the organisms. In the first case in which there was a subdural abscess the culture showed no growth; unfortunately in these cases anaerobic cultures were not made but that will be a "must" hereafter. In the second case also there was no growth from either the frontal sinus or from the subdural abscess. In the third case in which there was a left frontal lobe abscess the cultures revealed an unidentifiable organism, probably streptobacillus, sensitive to penicillin and to streptomycin. The cultures in many cases were made in cases in which large doses of antibiotics had been given before we saw the patients and apparently most of them had become sterile or, not having done anaerobic cultures, we did not pick up the presence of possible anaerobic bacilli.

With reference to Dr. Van Alyea's comments, I think these patients showed such virulent symptoms when first seen that antibiotics were definitely indicated early and vigorously. It is often difficult to find the organisms afterward. What we have found in extension from the ear pathology is that penicillin and aureomycin, when used early apparently kill off many of the gram-positive organ-

isms and leave very stubborn resistant gram-negative organisms such as proteus vulgaris and coliform organisms, which produce very stubborn infections.

The "Silent" Cholesteatoma

ROBERT HENNER

M. TAMARI

(Abstract)

This paper presents a group of five cases which at various stages or throughout the course of otologic disease showed no perforation of the drum or attic. Complicating symptoms such as vertigo, facial paralysis, deafness, or headaches were often the first symptoms. The conclusion is drawn that in the presence of obscure otologic symptoms a careful history and x-ray examination may reveal an otherwise unsuspected cholesteatoma of the attic.

DISCUSSION

DR. ROBERT LEWY: This is an excellent series of cases and I am sure we will all go away feeling better educated as a result of hearing them. However, unless I missed something, I do not see substantive proof offered for the statement Dr. Henner makes in his conclusions, that attic perforations, no matter how small, are always associated with cholesteatoma. This is contrary to my experience. Perhaps he can enlarge on this point and made it a little more clear to some of us who missed that point.

DR. SAMUEL SALINGER: The only comment I would offer is that I think the title of the paper is a misnomer. I do not believe the cholesteatomas were silent; I think they were extremely vocal, almost as vocal as the essayist! I think the difficulty was in recognizing the symptoms. So far as the connection between an attic perforation and cholesteatoma is concerned, I agree with Dr. Lewy; it does not always follow that cholesteatoma is present. One thing that might help in these cases, in which threatening symptoms supervene, would be more careful x-ray pictures in various projections. Owens of Los Angeles demonstrated a series of pictures whereby the details of the middle ear and attic could be shown very clearly and in many instances very small cholesteatomas could be identified. I am quite sure that his technique will demonstrate more than the conventional projections.

DR. FRANCIS LEDERER: Perhaps it is a misnomer to call these "silent" cholesteatomas; maybe the observers are silent in their thinking processes, because actually you see what happens when the otologist gets a case—it is no more a silent one. He discovers the true state of affairs. However, by and large, cholesteatomas are silent, and one has only to refer to the case treated as external otitis media eventually turn out to be what Dr. Henner and Dr. Tamari described in the first case.

It seems to me that the complacency that has overtaken us in medical treatment of otitis media has led us into the theory that antibiotics are good for everything, and has now produced an era of complacency that has permitted these cases to go on as long as they have. I would infer from the title, which I am sure is a catch title, that we are having brought to our attention again the thought that there are many serious cases of chronic suppurative otitis media that are unrecognized. I believe we will permit the essayists the privilege of using the word "silent" and defend it only on that ground.

DR. ROBERT HENNER (closing): Whenever one generalizes in science, certainly he is open to criticism, and our generalization that all small attic perforations had cholesteatomas is one from our experience. Just this week I operated on a case that could not be diagnosed with x-rays by any technic, because there was only a slit-like perforation in the lateral attic wall that admitted a fine perforating instrument such as is called a nerve probe. Attached to this in the attic was a small polyp, and under the microscope a beginning cholesteatoma was found.

The point we tried to make is that attic cholesteatoma should be suspected in all otologic complaints. About the title—the program omitted the quotation marks around the word "silent!"

EAR PROTECTORS—THE USE OF MILKWEED FLOSS AS A NEW MATERIAL IN EAR DEFENSE

RICHARD E. MARCUS, M.D.
L. JANE IRVINE, M.A.
GORDON STOWE, B.S.
AND
BUNNY RUBIN, M.A.

Using a pure tone audiometer (ADC 50E) in our sound controlled chamber, a calibration of the instrument was set up by testing the hearing of twelve people (24 ears) on each of the ears, on each of two successive days, by the same audiometrist, in the same room, and at different times during the day. The thresholds obtained on these ears were evaluated by statistical means and the reliability of the ADC 50E was then established in the testing situation.

Following the calibration, 30 persons between the ages of 20 and 45, chosen at random from a group of employes in our institution, were given complete otological histories and physical examinations, including the nose, mouth, teeth, tongue, pharynx, nasopharynx, auricles, external auditory canals, tympanic membranes, and visible ossicles. The sinuses were transilluminated, and clinical functional testing of the hearing, including tuning forks was done. No persons were used in the study in whom there was a history or demonstrable evidence of previous ear disease, or in whom there had been an exposure to damaging levels of noise.

Following the history and physical examinations, the persons were taken to the sound controlled chamber where an assistant explained that their hearing would first be tested without any insertions in the auditory canal, following which a pair each of three canal inserts would be put in the ears and new hearing tests obtained.

The inserts were chosen in previously determined random order (123, 231, 312, etc.). The inserts used were an ordinary plug of cotton pulled off the usual clinical swabbing, various sizes of the Mine Safety Appliance, and a stuffing of milkweed floss in a batiste covering.

After the testing was finished, each of the persons was asked if he could recall the various pairs of inserts and to describe for us, if he could recall them, which he liked the best.

It was concluded that:

(1) Insertion of (a) cotton swabbing, (b) milkweed floss covered with batiste, or (c) the manufactured Mine Safety Appliance device reduces auditory thresholds for pure tones. In each instance the reducation in auditory acuity is more pronounced in the higher frequencies than in the lower.

(2) The Mine Safety Appliance device reduces auditory acuity for pure tone to a greater degree in all frequencies than the cotton and the milkweed floss. Statistically, however, the milkweed floss and the Mine Safety Appliance device reduce acuity in the two highest frequencies (4098 and 8192) in the same degree.

(3) When used, coton and milkweed floss were the most predictable in their reduction of auditory acuity for pure tone, and the Mine Safety Appliance device the least predictable. This is believed due to the flexibility of the textile inserts.

(4) The Mine Safety Appliance device gave the most effective suppression in the speech range (500-2000 cps), the milkweed floss somewhat below, and the cotton inserts considerably below that.

(5) The subjects interviewed indicated that the milkweed floss inserts were the least uncomfortable of the three pairs tested.

(6) Milkweed floss should be further investigated as an ear defender in a field study involving larger groups of subjects in noisy environments of various types, and over longer periods of time.

DISCUSSION

DR. SHERMAN SHAPIRO: This is a much larger subject than was indicated by the title. I do not wish to discourage the essayist, but from my experience with safety engineers and insurance companies, I am doubtful whether he will get any large group of workers to submit to this ear plugging. That is one of the objections to ear defenders; the workers will not wear them. They do not feel right, often they interfere with conversation and the employes object to them.

So far as industrial noise is concerned, most industrial noise is in what the essayist characterized as the low frequencies. A 10 db reduction in noise, which is what one can expect to achieve from the

tests he made, is not enough. A loud industrial noise is 100 db or more and, when continued over a long period, is certainly damaging to the ear; I do not believe that that much reduction would do a preventive job.

There are number of these defenders of various types, some of which are objected to on the ground that they are unhygienic; if put in the ear they get dirty and cause external otitis. There is one on the market for which—believe it or not—it is claimed that it allows conversation to go through and when the noise gets too much a little diaphragm shuts the excessive noise off. I mention this as an example of some of the types shown.

A paper of this kind is worthwhile of course, but from what I understand of the subject the solution of industrial noise will not lie in defenders, but in proper acoustic engineering, and the redesigning of machines which will eliminate most of the noise. I do not believe you will ever get any large group of men to wear defenders continuously, even if they can be proven to be truly effective. Otherwise, I am much interested in the statistics and in the presentation of the paper.

DR. ROBERT HENNER: This is interesting to me because of my experience with a project in the air force in noise exposure. The spectrum of noise in an aircraft is over a large range, predominantly in the lower tones, and the damage is present usually with long exposure and is always in the range of 4096 to 8192, later encroaching on the speech range.

Lurie has shown in his experimental work, that with exposure to pure tones at 1000 cycles, the damage is not exactly at 1000 cycles but very often above that. This may show that cochlear localization of destruction of hair cells, as represented in high tones, may not be due to high tone damage.

Probably, the more protection one gets throughout the spectrum, the better it is for the individual—if you really want to protect him you must shut off the noise.

I am familiar with Dr. Shapiro's comment that psychologically it is almost impossible to get a large group of workers to use defenders. I think Dr. Marcus should be commended in his effort, because whatever protection can be afforded workers in industry is most important.

DR. RICHARD E. MARCUS (closing): It requires a coordinated education program participated in by the otologist, the industrial physician, and the employer, to acquaint workers with the nature

of the ear defender and the desirability of its use. We are just beginning to understand ear protection in industry, but the need is great and much can be expected to be done.

In reply to Dr. Shapiro, even a 10 db reduction may be effective for the purposes of ear protection. Many studies done in industry have indicated that there is a critical point at which noise becomes damaging to the cochlea, varying between 85 and 105 db in intensity, depending, of course, on the acoustical spectrum represented and the duration of exposure. Therefore, it is important to remember that the efficacy of an ear defender depends on its ability to reduce a potentially damaging noise to an intensity just below its critical point. A 120 db noise merely has to be reduced to 80 or 85 db in order to afford adequate cochlear protection. The noise is still loud, but is no longer damaging.

So far as acoustical engineering is concerned, I think we should take cognizance of the fact that whereas it may be important in industry, it will have little or no value to the military. There is no way known at the present time to do an effective acoustic engineering job on a 155 mm howitzer, and men still have to stand beside it or in front of it—at least underneath it—and are constantly caught by firing which they were not expecting. Protection of the cochlea will be afforded only by adequate local ear defense.

Dr. Henner mentioned the low tones. My present information indicates that frequencies below 500 have a less damaging potential to the cochlea than the frequencies above that range. Experimental work on the cochlea in which histologic examinations were carried out after exposure to discrete frequencies have shown damage localized a half octave or octave above the discrete frequency used. The overall impression would be that the higher frequencies are most responsible for cochlear damage, if exposure and intensity are sufficient. Therefore, ear defenders are most valuable if they reduce the intensity of the "highs."

Thorotrast Tumor of the Submaxillary Gland: Report of a Case

CARL D. MAKART

L. D. P., a woman aged 30, who had had a radiological examination with thorotrast six years previously, presented herself on April 17, 1952 because of a complaint of right submandibular swelling of eight to 12 weeks' duration which had become painful within the past

three weeks. She had a firm adherent mass 3.5 by 2.5 cc in size just below the horizontal portion of the mandible, somewhat anterior to the angle of the jaw. X-rays of both sides of the neck revealed a rather extensive area of diffuse opacity in the region of the submaxillary glands bilaterally. The carotid vessels were notably radiopaque.

At operation a densely fibrotic submaxillary gland was found, which was difficult to remove. As much of the gland was removed as was feasible. The histopathologic report was: densely fibrosed tissue adjacent to and in part involving the submaxillary gland with deposition of granular, translucent light brown material with no inflammatory reaction.

Summary: Although the use of radioactive thorium is being discontinued, one may anticipate the occasional appearance of an obscure tumor of the neck or mediastinum appearing long after cerebral arteriography. In the case cited, there was an interval of six years between radiographic study and development of a tumor. The diagnosis presents no great problem. However, one is inclined to be guarded as to the prognosis in these cases. Thorotrast is not without unwelcome sequelae. It produces parenchymatous alteration of perivascular tissue, according to some reports, and the incidence of a primary malignant sarcoma of the liver after twelve years has been reported.

DISCUSSION

Dr. B. J. Soboroff: This was a mass in the right submaxillary region, and although the films demonstrated extravasation of thorium and a dense picture on x-ray, still we could not rule out completely any other tumor in the submaxillary region. We explored the right upper neck through a transverse incision, exposing the submaxillary gland. The posterior portion of the gland was undefinable, actually merging with this dense fibrous tissue which extended up posteriorly to the region of the great vessels so that no clear line of demarcation could be made, and particularly exposure of the internal jugular vein could not be made. Since we were dissecting in an area where the internal jugular vein might be present, and we could not outline it clearly, we resected only a moderately large portion of the posterior part of the submaxillary gland. It did not appear to be tumor, it appeared to be dense fibrous tissue; it did not appear to be malignant; it was not friable and it did not bleed easily. We resected as much as we could safely and sent the tissue for pathologic examination. From the slide itself, the pathologist diagnosed a thorotrast granuloma.

DR. CARL D. MAKART (closing): Someone asked if we thought this was due to faulty technic in the administration of thorotrast. I do not think so, because there was 50 or 75 cc of the material used in the tumors first described; however, other investigators have used amounts as small as 20 to 40 cc and have had the same result, and tumors have occurred with even smaller amounts. It is believed that there is an affinity between the reticuloendithelial system and the thorotrast. In the present case, the trouble was only on the right side, although she still may have trouble on the other side. The physicists find this material to have half-life of 40 years, which is a long time.

EUGENE RICHARDS LEWIS, M.D.

1877-1953

It is with extreme regret that we note the passing of Eugene R. Lewis on March 18th at his home in Los Angeles.

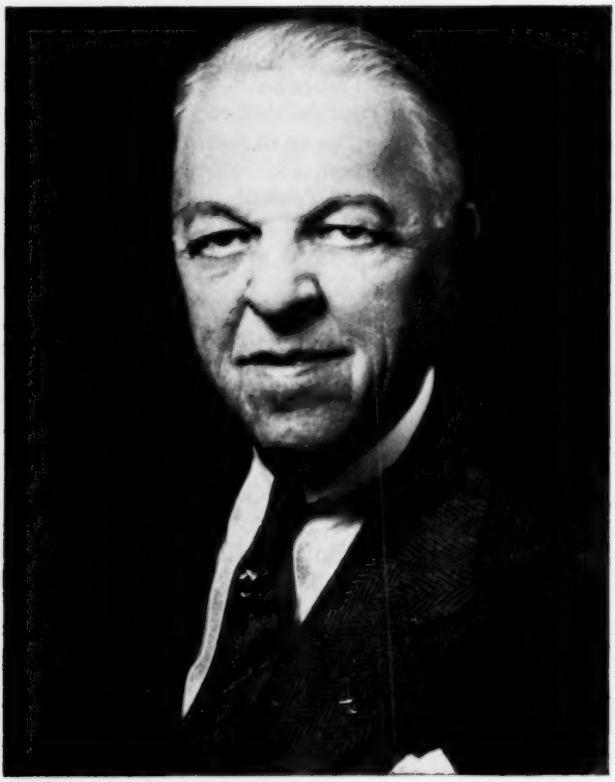
Dr. Lewis was born in Dubuque, Iowa May 14, 1877, the son of Dr. John S. Lewis and Harriet Richards Lewis. He received his medical education in the University of Pennsylvania from which he graduated in 1899. After serving as interne in Wilkes-Barre City Hospital and Mercy Hospital of Pittsburgh he returned to Dubuque to practice with his father. Recurrent otitis media induced him to leave Dubuque and prepare for the practice of diseases of the eye, ear, nose and throat.

After interning in Wills Eye Hospital of Philadelphia and Trinity Hospital of Milwaukee he continued his studies in London, Vienna, and Freiburg. In London he concentrated on pathology of the eye. On the continent he studied under Politzer, Neumann, Alexander, Hajek, and Barany.

Gene Lewis, as he preferred to be called, was a pioneer in the investigation of the vestibular apparatus. More than forty years ago he made the original observation that the anterior vertical canal of one side is in the same plane as the posterior vertical canal of the opposite side and that they are physiologically integrated.

Another important contribution from him was made when he advocated the addition of adrenalin to local anesthetic agents for the purpose of reducing bleeding during tonsillectomy. This inaugurated a technic which has long since become a universal procedure.

In 1917 when the Aviation Section of the U. S. Army was formed Dr. Lewis was appointed chief of the ear, nose, and throat department on the staff of General Theodore Lyster, the father of Aviation Medicine. He had before this been interested in the problem of how the eye, the ear, and the proprioceptive end organs work together in sensing motion. This appointment provided an opportunity for a comprehensive study by a group which included Isaac Jones, Lewis Fisher, and Henry Horn. Their work resulted in formulating the rules for the selection of pilots which are still included in the Aviation Manuel. He retired from the Reserve Corps of the Army with the rank of Colonel in 1919.



EUGENE RICHARDS LEWIS, M.D.
1877-1953

In 1920 he moved to Los Angeles and engaged in practice with former associates in the Army, General Theodore Lyster, Samuel Ingham, and Isaac Jones. He was appointed associate professor of Otolaryngology in the University of Southern California and attending physician to the Los Angeles General Hospital. These activities were continued until 1940 when an attack of coronary disease forced his retirement.

In addition to clinical practice he had many scientific and cultural interest. He was devoted to the basic sciences, particularly physics, biochemistry, and nutrition. Golf, music, sketching, and literature were his recreations. Those who knew him remember his brilliant conversations, ready wit, and delightful sense of humor.

In recognition of his value and achievements in otolaryngology he had been elected to membership in The American Otological Society, The American Laryngological Association, The American Laryngological, Rhinological, and Otological Society, The American Academy of Ophthalmology and Otolaryngology, and the Pacific Coast Oto-ophthalmological Society.

In 1904 Dr. Lewis married Elizabeth Bradley also of Dubuque, Iowa, born December 25th, 1879. He is survived by his widow, a sister, four children, and nine grandchildren.

D. H.

Abstracts of Current Articles

EAR

Modifications Introduced Into the Education of the Hard-of-Hearing by the Oscillograph.

Borel-Maisonny, M.: Revue de Laryngologie Otologie Rhinologie 73:459-467 (Sept.-Oct.) 1952.

A method of education is given in considerable detail in which hard of hearing children are taught speech perception and speech production. The salient feature is the cathode-ray oscillograph, which serves to give visual appreciation of the individual sounds and of phrases and sentences. Simultaneously the child receives tactual stimulation by placing his hands on the speaker's larynx and on his own larynx. Also, auditory stimulation is utilized by means of a hearing aid. At the same time the child watches the instructor's lips in the formation of sounds, and watches his own lips in a mirror. The author states that children of four or five years are old enough to begin such training and that great patience on the part of the instructor is needed in this educational work.

HARKINS.

The Capillaries of the Vestibular Membranous Labyrinth in the Guinea Pig.

Smith, Catherine A.: Laryngoscope 63:87-104 (Feb.) 1953.

This excellent study of the guinea pig labyrinth confirms the findings of previous workers regarding the arterial supply and venous drainage in the vestibular labyrinth, and extends the observations with a description of the capillary plexuses.

In this study, both the finding of a vein crossing the dome of the ampulla at right angles to the crista, draining the semicircular canal and ampulla and the observation of a vein coursing along the side were observed. A small part of the drainage occurs via these veins while the greater part of the canal is drained by the larger vein which usually is found leaving the utricular end of the canal.

The vascular pattern is similar in all of the neuroepithelial areas. The most dense capillary networks are the subepithelial plexuses found below the hair cell areas. The remainder of the membranous labyrinth is surrounded by a loose capillary network except along the convex surface of the semicircular canals and the free membranous covering of the saccule.

Observations in this series show that while the main route of vascular drainage from the labyrinth is through the vein of the cochlear aqueduct, the vein accompanying the endolymphatic duct is also of importance in the guinea pig. This may explain some of Perlman's observations on experimental occlusion of the vein of the cochlear aqueduct.

SENTURIA.

The Circulation of the Human Labyrinth.

Scuderi, R., and Del Bo, M.: Supplement, Archivo Italiano Di Otologia, Rinologia E Laringologia, pp. 90 (June) 1952.

The subject of this work is the distribution of the labyrinthine vessels in their capillary branches. The coloring method of Pickworth and Fazio was used on isolated parts of the labyrinth, as well as double coloration with the above method and with hematoxylin-eosin. For the study of the morphological and structural characteristics, rather thick sections were prepared, from temporals embedded in celloidin and very thin sections, from temporats embedded in celloidin and very thin sections, from temporats embedded in paraffin.

Although the vessels of the modiolus are characteristically winding it cannot be stated that they form real arterial glomerules, as is observed in other mammals: it was possible to observe that in man the vessels of the modiolus, even if they are of large range, show approximately the same characteristics as those of the capillaries, as their walls are very thin, composed of a single endolethial layer and of a reticular connective membrane which closes in its meshes numerous cells of an adventitious type (pericytes) and very scarce smooth muscle cells(in the larger vessels). No traces were found of elastic elements in the walls of the vessels of the modiolus.

From the principal parts of the cochlear artery itself, there occur secondary arterial ramifications, with a radical course, which leads upward towards the roof of the vestibular scala to the ligament and downward to the winding bone lamina and to the ganglial canal.

In the lamina spirale of man there exist:

1. Radial arterial vessels which present successive divisions in their flow from the modiolus up to the spiral crest.

2. Venous radial vessels, which collect the venous blood coming from the capillary nets and which flow into the vein of the spiral lamina and the spiral veins.

3. A spiral capillary net on the tympanic surface of the lamina.

4. A spiral capillary net of the crest and the vestibular surface.

5. A spiral vessel placed under the tunnel of the organ of Corti.

The presence of vessels has not been observed in Reissner's membrane, nor in the combed part of the basic membrane.

In the ligamentrum spirale and in the outside walls of the scala the vessel net is composed of:

1. Radial vessels (prevalently arterial) flowing in the vestibular scala.

2. Radial vessels (prevalently venous) which are found in the tympanic scala.

3. Arterio-venous anastomotic arches, situated deep in the thickness of the ligament and which form a direct connection between the radial vessels of the two scala.

4. Capillary net with a winding course of the stria vascularis.

5. Capillary net of winding prominence.

6. Capillary net with large meshes, interposed between the radial vessels of the vestibular scala (corresponding to the first net of Eichler).

7. Capillary net with large meshes between the vessels of the tympanic scala, not as evident as those mentioned above.

Radial arterial vessels reach the winding ganglion of Corti, which dissolve in a capillary net, diffused between the gangliar cells, from which a venous net starts, which, by means of collecting vessels, ends generally in the winding veins, and more rarely in the spiral lamina.

In the ampullae and in the semicircular canals, the arterial and venous vessels form a net of vessels which is in very close correspondence with the sensorial end points, and fairly rich also in the extrasensorial parts.

In the preparation of the utriculosaccular macula seen on the surface there can be focused, by proceeding from the outermost planes toward the endolymphatic cavity, the following elements:

1. The utricular and saccular arteries with their principal branches and the venous vessels that accompany them.

2. A large mesh net composed of the precapillary and capillary vessels, which are localized in the thickness of the lamina of the macula.

3. A subepithelial capillary net with very tight and irregular meshes, which very closely resemble the net of the stria.

There follows then a description of the circulation of the semicircular canals and the modiolus.

The existence of a rich sub-epithelial capillary net in the vascular stria and prominence, and in the maculae and the ampullar crests, may serve as confirmation of the secretory endolymphatic function as outlined by Fieandt and Saxen.

Production and resorption of perilymph is due to the subepithelial capillary net of the vestibular and tympanic scalae to the vessels enclosed in the bone cavities of the modiolus and above all to the rich net of vessles which surround the semicircular canals.

AUTHOR'S ABSTRACT.

NOSE

Terramycin in the Treatment of Rhinoscleroma.

Waeser, Von H., Schorndorf, M., and Schmidtmann, M.: Zeitschrift für Laryngologie Rhinologie Otologie 31:295-299 (June) 1952.

A case is presented of rhinoscleroma in a twenty-seven year old Hungarian woman. Clinically and histologically there were typical lesions of rhinoscleroma of the nasal mucous membrane, especially prominent on the inferior turbinates. Treatment by other measures, such as potassium iodide, x-ray therapy, local use of mineral oil, for more than a year had given no improvement. Two courses of Terramycin (250 milligrams at six hour intervals for sixteen doses) were given three months apart. There was gradual resolution of the lesions over the succeeding two months. At first there was an apparent increase in the local inflammatory reaction, an increase in the

number of foam cells of Mikulicz and notable phagocytic activity by the endothelial cells. This gradually subsided and the mucosa took on a normal character, without the dense scar tissue usually seen. Very good photomicrographs of tissue taken for biopsy during various stages of the case are included in the original paper.

HARKINS.

Fibroma of the Frontal Sinus.

Schwab, Werner: Zeitschrift für Laryngologie Rhinologie und Otologie 32:23-27 (Jan.) 1953.

The author reports a case of a true fibroma of the frontal sinus. The patient was a forty-eight year old woman with a history of displacement of the left orbit downward and outward of about one year's duration, with only a vague sense of pressure in the region of the left orbit and frontal sinus. The nasal cavities were clear. There was a palpable swelling at the inner portion of the left orbital roof. Roentgen examination showed enlargement of the left frontal sinus with only mild generalized clouding of this cavity. The bony outlines appeared sharp. The patient was operated upon by the external frontal approach. The medial part of the floor of the left frontal sinus was paper-thin displaced downward by the tumor mass. The entire sinus was found to be filled with this fleshy tumor, which was attached to the anterior wall. The posterior wall of the sinus had been completely eroded, exposing the dura, and a broad extension of the tumor had pushed its way into the ethmoid cells. The entire neoplasm was removed and the incision closed.

Histologically the tumor consisted of masses of rather cellular fibrous tissue, arranged in broad bundles. It was classified as a periosteal fibroma. The surface was smooth, lobulated and covered with epithelium. There was no evidence of new bone formation. Such a fibroma within one of the nasal sinuses is an uncommon condition. In the differential diagnosis, mucocele and osteoma of the frontal sinus were considered and discussed.

HARKINS.

LARYNX

Contributions to the Treatment of Stenosis of the Larynx.

Guns, P.: Revue de Laryngologie Otologie Rhinologie 73:209-216 (May-June) 1952.

The author reports two interesting cases of laryngeal stenosis.

In the first case there was complete stenosis, the result of an old injury to the larynx. In this instance the larynx was laid open by a median thyrotomy and all scar tissue dissected out from the interior. An acrylic mold was then covered with a Thiersch skin graft and sutured into the larynx, wihch was then closed. The acrylic mold also served as an airway. It was left in place for three months and then removed by direct laryngoscopy after cutting the sutures. After four years the airway remains excellent, but the voice is husky.

In the second case there was tuberculosis of the larynx, as well as of the lung. The patient was kept on streptomycin for nearly a year. The larynx healed with an adherent scar between the anterior portions of the vocal cords, resulting in aphonia and respiratory difficulty. After several trials with simple methods proved unsuccessful, the author adopted the method of McNaught. The lower portion of the larynx was opened in the midline, only high enough to expose the cords. The scar was sectioned in the midline with the electric cautery. Then a small sheet of tantalum was introduced between the anterior ends of the vocal cords and sutured in place. This was left in for two months, and was easily removed through a small external incision. The result has been good: respiration was restored to normal and the voice has been fairly strong, but husky.

HARKINS.

Late Development of Laryngeal and Pharyngeal Carcinoma in Previously Irradiated Areas.

Holinger, Paul H., and Rabbett, William F.: Laryngoscope 63:105-112 (Feb.) 1953.

The authors report from the literature ten cases of carcinoma of the deep structures of the neck that occurred following irradiation of the cervical region and add three patients of their own who showed malignant lesions of the hypopharynx or larynx in direct line of the previously administered x-ray therapy.

In all cases irradiation had been given 16 to 32 years before the development of the neoplasm. In the authors' three cases, the tumor had its origin on the side previously irradiated. It is interesting and significant that the ratio of females to males was three to one, a reversal of the usual incidence for this kind of tumor.

It is conjectured that more of these cases will be seen in the near future as a result of the frequency with which irradiation therapy was prescribed for benign conditions several decades ago.

SENTURIA.

ESOPHAGUS

Statistical Data Concerning Malignant Tumors of the Esophagus.

Frühling, M. L., and Wild, C. H.: Les Annales d'Oto-Laryngologie 69:400-419, 1952.

In a statistical and pathological analysis of a series of 598 cases of malignant tumors of the esophagus (547 esophageal cancers studied by biopsy or at operation, and 51 cases diagnosed at autopsy) the authors drew the following conclusions:

The frequency of the mortality from cancer of the esophagus has not varied in the past two decades; cancer of the esophagus accounts for 84 per cent of death from all causes and approximately 3 per cent of deaths from cancer. The age incidence has increased slightly in the past twenty years from 60.6 years to 61.5 years. Histologically, by far the most frequent type is the squamous epiphelioma. Less common are glandular carcinoma and cylindrical epitheloma, arising either at the cardia or from misplaced islands of gastric epithelium in the esophagus. In this series primary sarcomata were rare, constituting 1.5 per cent of the total. Secondary carcinomata of the esophagus, constituting 2 per cent of the total, arose either in the stomach or in the bronchi. A full discussion of the histological patterns of these various lesions is given.

HARKINS.

MISCELLANEOUS

Fibromas and Angiomas of the Rhinopharynx.

Alcaino, Alfredo: Rev. de Otorrinolaring. 12:42, 1952.

In this paper the author discusses the origin of fibromatous and angiomatous lesions of the nasopharynx and presents his original surgical procedure for removal of them.

Because of the characteristic pedunculation of these tumors he believes that they originate in the nose rather than the vault of the pharynx and are forced into the pharynx by their own growth. This produces the pedunculation which has so generally associated them with the pharynx.

To overcome the difficulties usually encountered in removing them completely the author makes an exposure as follows:

1. The septal mucosa is incised from the edge of the quadrangular cartilage down to the middle of the floor of the nose.

2. After elevating the perichondrium and periosteum the cartilage and perpendicular plate are fractured away from the side of the growth.

3. The middle and lower turbinates of both sides are fractured and displaced laterally. The additional space provided will enable the operator to view the tumor and remove it completely.

4. The operation is completed by replacing the septum and the turbinates into the normal position.

HIGBEE.

Fracture of the Malar Bone and Methods of Treatment.

Stuteville, O. H., and Levignac, J.: Les Annales d'Oto-Laryngologie 69:294-310, 1952.

The entire subject of fractures of the malar bone is reviewed and the authors' own experience in treating these injuries is discussed. Symptoms and signs are describe in detail, particular emphasis being placed on lowering of the orbit, with diplopia, restriction in opening the mouth, flattening of the prominence of the cheek, and anesthesia of the face. As to treatment the authors do not feel that it can be

limited to any one form. In some cases they use a small incision in the temporal fossa after the method of Gillies. In other cases they make an incision in the inferior palpebral fold to expose the inferior bony margin of the orbit for direct manipulation and reduction, or they may use a short vertical incision just lateral to the eyebrow to expose the bone at the zygomaticofrontal suture. In some instances they open the antrum by the Caldwell-Luc technic and elevate the fragments by this approach, packing the antrum for four or five days thereafter. A combination of two or more of these approaches has been used successfully. In early and simple cases reduction alone has been sufficient. In cases of long standing or those with comminution, wiring the fragments together has been necessary.

This article is of particular interest because it is an original contribution in this French journal, but originates in the Department of Maxillo-Facial Surgery of the Cook County Hospital in Chicago. The authors state that malar fractures are frequent in that hospital, a fact which they attribute to numerous street fights, drunken tavern brawls, robberies, as well as traffic accidents, on the West Side of Chicago.

Total Excision of the Submaxillary Gland and Wharton's Duct by an Intraoral Method.

Bourguet, J., and Branchu, J. L.: Les Annales d'Oto-Laryngologie 69:194-199, 1952.

The authors describe in detail their method of excision of the submaxillary salivary gland through an intraoral incision. They have used it in three cases having calculi in the gland and duct. They give as advantages of this method the fact that the gland and its duct can be excised totally in one piece and that there is no external scar. The authors review the anatomy of this region in detail. The operative procedure is described. An incision is made in the mucosa from the frenulum as far back as the third molar tooth. A sound is passed into the submaxillary duct, which serves as a guide in dissecting back into the submaxillary fossa and in crossing over the lingual nerve, which is spared. The dissection posteriorly into the depths of the fossa is along the outer aspect of the hyoglossus muscle. The mylohyoid muscle acts as somewhat of a barrier. By gentle traction and blunt dissection the operator is able to free and deliver the anterior lower pole of the gland from below this muscle. The authors caution against injury to the anterior facial artery and vein in dissecting free the gland. In two of their cases they encountered direct

branches of the artery, which they clamped and ligated. The wound is not closed, but is packed with iodoform gauze, which is left in for 48 hours.

HARKINS.

Advances in the Use of Antibiotic Substances.

Bunn, Paul A.: Laryngoscope 63:128-136 (Feb.) 1953.

The author reviews and comments on the advances in the use of antibacterial substances. Reference is made to the great activity of stilbamidine, administered parenterally against blastomycosis. Now this formerly fatally progressive fungus disease can be controlled and probably cured.

A second important advance is the development of isonicotinic acid hydrazide, a chemical which is not of biologic origin. It is cheap, innocuous and active in the human and exerts a bactericidal effect upon the growth of tubercle bacilli.

Penicillin G diethyl amino-ethyl ester hydroiodide has two unusual pharmacological advantages. It is unique in that it is the first preparation of penicillin to penetrate the blood-brain barrier and therefore is a valuable agent in meningitis. Secondly, but of less importance, is the fact that it is found in significant concentrations in the bronchial secretions.

Dibenzyl ethylene diamine dipenicillin is another penicillin ester which is released at a very slow rate and will give blood levels extending well over seven days and may have an important place in long term prophylaxis.

The author expresses strong feelings regarding the use of combinations of antimicrobial substances. He insists that, with few exceptions, a single drug, used correctly, is just as effective as a combination of drugs. The vast majority of infections do not require polyantimicrobic therapy while there are clear-cut contraindications to the simultaneous use of two or more agents.

He is also much opposed to the topical use of antibiotics and advises systemic administration so that it reaches the site of infection by way of the blood stream, thus reaching the base or periphery of the inflammation where the most active reproduction of organisms is occurring. Attention is again called to the necessity for continuing adequate therapy for a minimum of five days if non-purulent complications are to be avoided.

SENTURIA.

Notices

AMERICAN LARYNGOLOGICAL ASSOCIATION

CASSELBERRY PRIZE

A sufficient sum having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award. Theses must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia, Pa., before March 1, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

October 6-10, 1953, in Chicago, Ill., at the Palmer House.

DEAN M. LIERLE, M.D., Secy.
University of Iowa
Iowa City, Iowa.

ACADEMY HOME STUDY COURSES

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

PAN-AMERICAN CONGRESS

Fourth Pan-American Congress of Oto-rhino-laryngology and Broncho-esophagology, Mexico City, January 1954. Dr. Ricardo Tapia Acuna, President, Dr. Chevalier L. Jackson, Executive Secretary, 1901 Walnut St., Philadelphia 3, Pa.

UNIVERSITY OF ILLINOIS

A course in Bronchoesophagology is to be given by the University of Illinois College of Medicine October 19-31, 1953, under the direction of Dr. Paul H. Holinger.

Please address the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

INSTITUTE ON ESOPHAGEAL SPEECH

An Institute on teaching and improving esophageal speech to be held in Cleveland, Ohio, August 10 through the 16th at The Cleveland Hearing and Speech Center is being sponsored by the American Cancer Society, National Cancer Institute of Institutes of Health, the Office of Vocational Rehabilitation, the Cleveland Otolaryngological Society, the Cleveland Academy of Medicine and the Western Reserve University School of Medicine.

Non-speaking laryngectomized persons are invited to attend without charge.

Surgeons, speech pathologists and lay persons are invited to attend the sessions. Limited registration is necessary. Application must be mailed by July 15 to Warren H. Gardner, Ph.D., Program Chairman, 11206 Euclid Ave., Cleveland 6, Ohio.

UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology, divided into two sections:

A. Basic Section, September 21 through 26, 1953, devoted to surgical anatomy and dissection of the head and neck, and histopathology of the ear, nose and throat, under the direction of Dr. M. F. Snitman.

B. Clinical Section, September 28 through October 3, 1953, consisting of lectures and panel discussions, with group participation of otolaryngological problems and current trends in medical and surgical management.

Registration will be limited. Application for attendance at one or both sections will be optional. For information write to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

(As of June 1, 1953)

Audicon Models 400, 415, 530 and 615
Audiotone Model 11
Audivox Models Super 67 and 70
Aurex Models L and M

Beltone Mono-Pac Model "Lyric"
Beltone Mono-Pac Model M
Beltone Mono-Pac Model "Rhapsody"
Beltone Symphonette Model

Cleartone Models 500 and 700
Cleartone Regency Model

Dahlberg Model D-1
Dahlberg Junior Model D-2
Dahlberg Model D-3
Dahlberg Model D-4
Dysonic Model 1

Fortiphone Models 20A and 21C

Gem Models V-35 and V-60
Goldentone Models 25, 69 and 97

Maico Model J
Maico Quiet Ear Models G and H
Maico Top Secret Model L
Moico UE Atomeer
Mears (Crystal and Magnetic) Aurophone Model 200
Micronic Model 303
Micronic Model Mercury
Micronic Star Model
Microtone Classic Model T-9
Microtone Models T-10, T-612 and 45

National Cub Model (C)
National Model D (Duplex)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506
Normatone Model C

Otarion Model E-4
Otarion Models F-1, F-2 and F-3
Otarion Models G-2 and G-3
Otarion Model H-1

Paravox Model D (Top-Twin-Tone)
Paravox Model J (Tiny-Myte)
Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Radioear All-Magnetic Model 55
Radioear Permo-Magnetic (Multi-power)
Radioear Model 62 Starlet
Radioear Model 72
Radioear Model 82 (Zephyr)
Rochester Models R-1 and R-2

Silvertone Models J-92 and P-15
Solo-Pak Model 99
Sonotone Models 900, 910, 920, 925, 940 and 966
Super-Fonic Hearing Aid

Televox Model E
Telex Models 97, 99, 200, 300-B, 400, 500, 952, 953 and 1700
Tonamic Model 50
Tonemaster Model Royal
Tonemaster Cameo Model

Unex Midget Models 95 and 110
Unex Models 200 and 230

Vacolite Models J and J-2

Western Electric Models 65 and 66

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal
Zenith "Regent"

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

SEMI-PORTABLE HEARING AIDS

Ambco Hearing Amplifier (table model)

Aurex Semi-Portable

Precision Table Hearing Aid

Sonotone Professional Table Set Model 50

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Blvd., Chicago 11, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Board of Otolaryngology

President: Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

American Broncho-Esophagological Association

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

American Laryngological Association

President: Dr. Gordon F. Harkness, 2410 River Drive, Davenport, Iowa.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Boston, Mass., May 20 and 21, 1954.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis LeJeune, Prytania and Olive Sts., New Orleans, La.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: New Orleans, La., April 28-30, 1953.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

American Otological Society

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary-Treasurer: Dr. John R. Lindsay, 950 East 59th St., Chicago 37, Ill.
Meeting: Boston, Mass., May 16 and 17, 1954.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: Kenneth L. Craft, M.D., 23 East Ohio Street, Indianapolis, Indiana.
President-Elect: Albert D. Ruedemann, M.D., 1633 David Whitney Building, Detroit 26, Michigan.
Secretary-Treasurer: Michael H. Barone, M.D., 468 Delaware Avenue, Buffalo 2, New York.
Meeting: Chicago, Illinois on October 16, 1953.

International Bronchoesophagological Society

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.
Meeting: Second International Congress of Bronchoesophagology, June 15th and 16th, 1953, Albert Plage, Le Zoute, Belgium.

Pacific Coast Oto-Ophthalmological Society

President: Leland G. Hunnicutt, M.D., 98 N. Madison Ave., Pasadena 1, Calif.
Secretary-Treasurer: John F. Tolan, M.D., 3419 47th Ave., N.E., Seattle 5, Wash.
Meeting: Honolulu, 1954.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso, Montevideo.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.
Meeting: Fourth Congress of Oto-rhino-laryngology and Broncho-esophagology.
President: Dr. Ricardo Tapia Acuña, Mexico City.
General Secretary: Dr. Maximo García Castaneda.
Time and Place: February 28th to March 4th, 1954, Mexico City.